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TRACHEOTOMY IN CASES OF ACUTE OBSTRUCTIVE NONDIPHTHERITIC INFECTIONS OF LARYNX, TRACHEA AND BRONCHI

GENERAL STUDY, WITH AN ANALYSIS OF ONE HUNDRED AND TWENTY-SIX CONSECUTIVE CASES OCCURRING DURING THE PAST DECADE

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The purpose of this paper is to review my general experience with cases of acute obstructive nondiphtheritic laryngitis, laryngotracheitis and laryngotracheobronchitis requiring tracheotomy.[1] Although the disease is, fortunately, relatively uncommon, the fact that the mortality rate has ranged from 50 to 100 per cent, depending on the virulence of the infection and the type of treatment, makes its study important. In passing, it might be mentioned that in 1929 the condition in 60 per cent of all patients admitted to the Willard Parker Hospital for acute laryngeal obstruction was due to diphtheria, whereas in 1939 in only 10 per cent was the obstruction caused by infection with the diphtheria bacillus. Thus, the nondiphtheritic infections of the larynx, trachea and bronchi have assumed a much greater numerical importance.

The plan of this study is (1) to present a general statistical survey of the past decade; (2) to cite unusual occurrences at the time of operation; (3) to warn of various dangers following operation which are frequently overlooked; (4) to outline detailed postoperative care, including complications and problems connected with decannulation, and (5) to analyze the sequelae found from one to ten years after the illness in patients who had intubation or tracheotomy or both. A comprehensive discussion of tension pneumothorax and mediastinal emphysema

From the Willard Parker Hospital, New York.

1. Neffson, A. H., and Wishik, S. M.: Acute Infectious Croup: A General Study of Acute Obstructive Infections of the Larynx, Trachea and Bronchi with an Analysis of Seven Hundred and Twenty-Seven Cases, J. Pediat. **5**:433 (Oct.); 617 (Nov.); 776 (Dec.) 1934. These articles give a comprehensive review and a discussion of the treatment.

has been given elsewhere.[2] Knowledge of the straightforward procedure of tracheotomy is taken for granted.

Cases of such conditions as diphtheria, blood dyscrasias, cervical abscess and neoplasms, Ludwig's angina, retropharyngeal abscess and large, pendulous obstructive tonsils and cases in which tracheotomy was performed just before admission but decannulation was done on admission are not included in this study. However, a number of cases of acute obstructive laryngitis occurring as a complication of scarlet fever, measles and varicella have been included because they present problems similar to those in cases of nonspecific infection of the respiratory tract.

With the striking decrease in the incidence of diphtheria during the past decade, the practice of intubation has gradually fallen into desuetude, and the art has largely been lost. Since the work reported on here was done in one of the few remaining hospitals in the country where intubation is still employed by specially trained residents, the bearing of this procedure on the subject under consideration will be discussed. Thus, the question of tracheotomy versus intubation, which usually arises at any general pediatric-laryngologic discussion of diphtheria, will also be considered here.

INCIDENCE

A. Frequency.—During the past decade, from 1931 to 1940, 1,360 cases of nondiphtheritic infectious obstruction of the larynx, trachea and bronchi occurred in the Willard Parker Hospital. Of these, 1,113 were treated by nonsurgical means, e. g., with rest, fluids, vigorous antipyretic measures, steam inhalations, oxygen, specific serums, antigens and chemotherapy, as well as direct laryngoscopy with laryngeal and laryngotracheal aspiration in a large number of cases.[1] Intubation, in addition to aspiration, was required for relief of the obstruction in 121 cases (8.9 per cent). Tracheotomy was resorted to in 126 cases (9.2 per cent). Of the 126 cases in which tracheotomy was performed, intubation had previously been done in 93 (74 per cent), while in 33 cases (26 per cent) intubation was deemed inadvisable or inadequate (table 2).

Table 1 shows the number of all cases of acute nondiphtheritic obstructive laryngitis, laryngotracheitis and laryngotracheobronchitis in the Willard Parker Hospital, the incidences of intubation and tracheotomy, together with the mortality rates for these procedures for each respective year, the first five years, the last five years, the first seven years, the last three years and the entire decade. The data for each year were tabulated from July 1 to June 30 in order to obtain a more accurate picture of the yearly epidemics.

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2. Neffson, A. H.: Tension Pneumothorax and Mediastinal Emphysema in Acute Obstructive Infections of the Larynx, Trachea and Bronchi Requiring Tracheotomy: A General Study with an Analysis of Seventeen Cases in a Series of One Hundred and Twenty-Six Tracheotomies During the Past Decade, Arch. Otolaryng., to be published.

Examination of these statistics reveals several interesting points.

1. In the first half of 1931 the incidence of tracheotomy was 1.4 per cent and that of intubation 5.5 per cent, and the general mortality rate was 16 per cent—9 deaths occurring in patients who came in a moribund condition or died before intubation or tracheotomy could be performed

TABLE 1.—*Frequency and Mortality Rate of Tracheotomy and Intubation at the Willard Parker Hospital*

	Tracheotomies (With Intubation and Without Intubation)				Intubations (No Tracheotomy)				All Cases of Acute Obstructive Laryngitis		
Year	Number	Deaths	Frequency, %	Mortality, %	Number	Deaths	Frequency, %	Mortality, %	Number	Deaths	Mortality, %
1931 (1st half)	1	1	1.4	100	4	2	5.5	50	73	12	16
1931-1932	8	5	5	62.5	22	12	13	54	166	18	11
1932-1933	6	4	3.3	66.6	17	8	10	47	178	24	13.5
1933-1934	6	3	4	50	5	0	3.4	..	145	6	4.1
1934-1935	8	4	8	50	5	1	5	20	104	7	6.7
1935-1936	5	1	3.3	20	14	9	9.3	64	150	14	9.3
1936-1937	11	7	10	63.6	8	4	7	50	117	12	10.3
1937-1938	15	3	15	20	24	10	23.5	42	102	14	14
1938-1939	24	7	25	29	10	1	10.6	10	94	8	8.5
1939-1940	37	9	19.4	24	12	3	6.3	25	190	13	7
1940 (2d half)	5	..	12	....	..	..	..	..	41	1	2.4
Grand total 1931-1940	126	44	9.2	35	121	50	8.9	41.3	1,360	129	9.5
First 5 yr. 1931-1935	32	18	4.3	56	60	25	8	43	744	73	9.8
Second 5 yr. 1936-1940	94	26	15.2	28	62	25	10	40	616	56	9
First 7 yr. 1931-1937	52	26	5.3	50	85	40	8.7	47	978	97	10
Last 3 yr. 1938-1940	74	18	19.4	24.3	36	10	9.4	28	382	32	8.5

TABLE 2.—*Comparison of Cases of Tracheotomy with Intubation and Without Intubaton at the Willard Parker Hospital*

Type	With Intubation	Without Intubation
Number	93	33
Deaths	28	16
Mortality, per cent	30	48.5

or for whom neither intubation nor tracheotomy was thought to be indicated or advisable. In the last half of 1940, although the incidence of tracheotomy was 12 per cent, the general mortality rate was only 2.4 per cent, and the single death was sudden, from supraglottic edema, before tracheotomy could be done.[3]

3. Neffson, A. H.: Supraglottic Laryngeal Edema: Characteristic Respiratory Sounds as an Aid in Diagnosis, Ann. Otol., Rhin. & Laryng. **46**:1065 (Dec.) 1937.

2. In the first five years the incidence of tracheotomy was 4.3 per cent, the mortality rate for tracheotomy was 56 per cent and the general mortality rate was 9.8 per cent. During the last three years the incidence of tracheotomy was 19.4 per cent, almost five times as high; the mortality rate for tracheotomy was 24.3 per cent, or less than one-half, and the general mortality rate was 8.5 per cent, the last reduction not being statistically significant. During the same periods the incidence of intubation alone hardly varied (from 8 to 9.4 per cent), but the mortality rate for intubation dropped from 43 to 28 per cent.

(*a*) The explanation for the increase in tracheotomies lies in the general tendency of the resident and the laryngologic staff to perform tracheotomy, as well as intubation, more often and sooner than heretofore. (The combined incidence of tracheotomy and intubation was 14 per cent during the first seven years and 28.8 per cent during the last three years [table 4]). In some cases the patient would undoubtedly have recovered with intubation alone, or even with nonsurgical treatment, but the more frequent performance of tracheotomy does not seem to have resulted in any higher general mortality rate. On the contrary, the mortality was even lower, though the drop is not statistically significant.

(*b*) The lowering of the mortality rate for tracheotomy was to be expected, since some of the patients were not as dangerously sick as in the past and might have recovered without operation. The concomitant reduction, from 43 to 28 per cent, in the mortality rate in the cases in which intubation was employed could be explained in the same manner, since intubation, and later tracheotomy, was performed in a greater number of cases and the cases were then grouped under "tracheotomy." However, I believe the reduction in the mortality rates for tracheotomy and intubation must be ascribed not only to the less rigid indications for mechanical intervention, but to the better postoperative care and the introduction of chemotherapy. (The role of chemotherapy will be considered later in the paper.)

3. Although Brennemann and associates [4] and others have stated that the general virulence of an epidemic is roughly proportionate to the number of cases of the disease, a glance at table 1 tends to show that the numerical incidence and the mortality rates do not necessarily run hand in hand. For example, in 1934-1935 there were 104 cases and the mortality rate was 6.7 per cent, whereas in 1937-1938 there were 102 cases and the mortality rate was 14 per cent. Again, in 1933-1934, with a higher incidence of 145 cases, the mortality rate was the lowest of any full year, only 4.1 per cent, and in 1935-1936, with practically the same incidence, 150 cases, the mortality rate was 9.3 per cent, or

4. Brennemann, J.; Clifton, W. M.; Frank, A., and Holinger, P. H.: Acute Laryngotracheobronchitis, Am. J. Dis. Child. **55**:667 (April) 1938.

twice that of the preceding year. In 1938-1939, although there were the fewest admissions, only 94, the mortality rate was 8.5 per cent, whereas in 1939-1940, with the most admissions, 190, the mortality rate was only 7 per cent. Moreover, the incidence of tracheotomy did not parallel the general mortality rate; e. g., in 1932-1933 tracheotomy was performed in only 3.3 per cent of the 178 cases and the general mortality

TABLE 3.—*Frequency and Mortality Rates of Tracheotomy and Intubation at the Kingston Avenue Hospital* *

Year	Tracheotomies With and Without Intubation				Intubations (No Tracheotomy)				All Cases of Acute Obstructive Laryngitis		
	Number	Deaths	Frequency, %	Mortality, %	Number	Deaths	Frequency, %	Mortality, %	Number	Deaths	Mortality, %
1931	6	2	5.3	33.3	23	11	20	47.8	113	21	18.5
1932	2	2	1.4	100	21	10	14.8	47.6	142	17	11.9
1933	2	..	1.4	....	23	2	16.4	8.6	140	12	8.5
1934	3	3	2.6	100	14	4	12.4	28.5	113	10	8.8
1935	7	3	4.2	42.8	32	9	20	28.1	164	14	8.5
1936	1	..	1	....	12	3	12	25	102	5	4.9
1937	3	..	2.6	....	17	5	15	29.4	113	6	5.3
1938	5	3	4.1	60	6	1	5	16.6	121	7	5.7
1939	8	5	62	62.5	7	3	5.4	42.8	129	13	10
1940	17	9	11.3	52.8	5	3	3.3	60	150	16	10.6
Grand total 1931-1940	54	27	4.2	50	160	51	12	32	1,287	121	9.4
First 7 yr. 1931-1937	24	10	2.7	41	142	44	16	31	887	85	9.6
Last 3 yr. 1938-1940	30	17	7.5	57	18	7	4.5	39	400	36	9

* These data were taken from the monthly reports of the Kingston Avenue Hospital.

TABLE 4.—*Comparison of Frequency and Mortality Rates for Tracheotomy and Intubation at the Willard Parker and the Kingston Avenue Hospital*

Year	1931-1940		1931-1937		1938-1940	
Hospital	Willard Parker	Kingston Avenue	Willard Parker	Kingston Avenue	Willard Parker	Kingston Avenue
Incidence of acute obstructive laryngitis	1,360	1,287	978	887	382	400
General mortality rate, %	9.5	9.4	10	9.6	8.5	9
Incidence of tracheotomy, %	9.2	4.2	5.3	2.7	19.4	7.5
Mortality rate for tracheotomy, %	35	50	50	41	24.3	57
Incidence of intubation, %	8.9	12	8.7	16	9.4	4.5
Mortality rate for intubation, %	41.3	32	47	31	28	39
Combined incidence of tracheotomy and intubation, %	18.1	16.2	14	18.7	28.8	12
Combined mortality rates for tracheotomy and intubation, %	38	41	48.5	36	26	48

rate was 13.5 per cent, whereas in 1939-1940 tracheotomy was done in 19.4 per cent of the 190 cases and the general mortality rate was only 7 per cent. It thus becomes evident that conclusions as to the virulence of an epidemic either from the incidence of the cases or from the incidence of tracheotomy are dangerous.

For purposes of comparison, table 3 shows the number of all cases of acute nondiphtheritic obstructive laryngitis, laryngotracheitis and laryngotracheobronchitis occurring in the Kingston Avenue Hospital, Brooklyn. (This hospital is of the same type as the Willard Parker Hospital—for contagious diseases.) The table also shows the incidences and mortality rates during the past decade for cases in which intubation and tracheotomy were employed. Table 4 compares more specifically the frequencies and mortality rates for tracheotomy and those for intubation, as well as the combined incidences and mortality rates for tracheotomy and intubation at the two institutions during similar periods. It will be noted that the incidences of acute obstructive laryngitis during the entire decade, and also during the lesser periods, were almost the same in the two hospitals. Moreover, the general mortality rates varied only by 0.1 to 0.5 per cent. In both institutions the incidence of tracheotomy increased during the last three years as compared with that during the first seven years—in the Willard Parker Hospital from 5.3 to 19.4 per cent and in the Kingston Avenue Hospital from 2.7 to 7.5 per cent. With the increase in the incidence of tracheotomy, the mortality rate for tracheotomy fell from 50 to 24.3 per cent at the Willard Parker Hospital, but rose from 41 to 57 per cent at the Kingston Avenue Hospital. (It should be mentioned that during the last three years intubation fell into disfavor at the Kingston Avenue Hospital, the incidence of this procedure falling from 16 per cent for the first seven years to 4.5 per cent for the last three years.)

An interesting observation is that the combined incidences of tracheotomy and intubation fell at the Kingston Avenue Hospital from 18.7 per cent during the first seven years to 12 per cent during the last three years, but rose at the Willard Parker Hospital from 14 to 28.8 per cent during the same period. Furthermore, if the combined mortality rates for tracheotomy and intubation are compared with the combined incidences of tracheotomy and intubation at both hospitals, a remarkable relationship is seen to exist; namely, with the lowest rates of intervention there are the highest mortality rates, and with an increase in the rate of intervention there is an almost mathematically proportionate decrease in the mortality rate. Thus, when the combined incidence of tracheotomy and intubation was only 12 per cent, the combined mortality rate was 48 per cent; when it was 16 per cent, the mortality rate was 41 per cent; when it was 18 per cent, the mortality rate was 38 per cent; when it was 18.7 per cent, the mortality rate was 36 per cent, and when it was 28.8 per cent, the mortality rate was only 26 per cent. Moreover, at the same time the general mortality rates for all cases of acute obstructive laryngitis hardly varied.

Thus it would appear that the amount of operative intervention has little effect on the general mortality rate, provided the postoperative care

is good. However, from clinical experience, I should say that the less operative intervention the better so far as the general morbidity is concerned. On the other hand, good judgment as to how long one can safely wait before resorting to operation is difficult to acquire, and therefore early intervention is generally wiser, provided the postoperative care is good.

B. Age.—The age range for the 126 tracheotomized patients was from 5 months to 47 years. The greatest number were between the ages of 1 and 2 years. The curve showing this distribution (fig. 1) follows the general curve noted in a previous paper.[1]

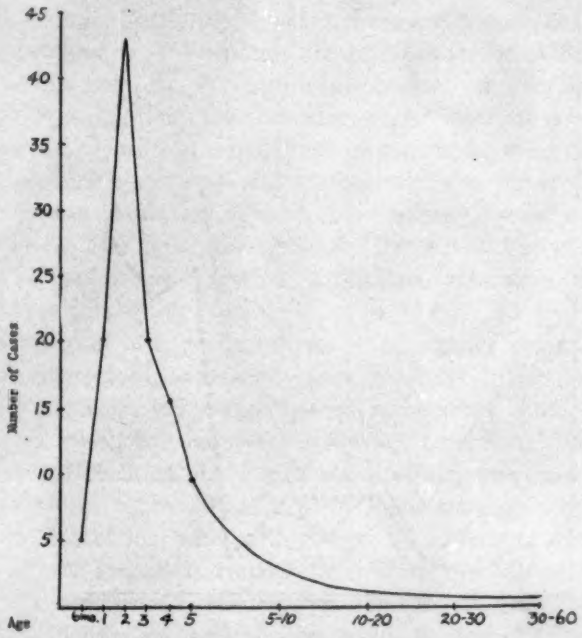

Fig. 1.—Age incidence of 126 tracheotomized patients.

C. Sex.—Of the 126 patients, 88 were males (70 per cent) and 38 females (30 per cent). This proportion holds close to the incidence of males and females in the entire group of patients with acute obstructive laryngitis.[1]

BACTERIOLOGY

Examination of figure 2 shows a remarkable increase since 1938 in the incidence of Staphylococcus aureus, chiefly in pure culture. During the same period there was little change in the incidence of Streptococcus haemolyticus or Streptococcus viridans. This increase in the occurrence of Staph. aureus was likewise noted in the general

run of cases of acute obstructive laryngitis (without tracheotomy) at the Willard Parker Hospital, throughout New York city and in the surrounding region.

In an analysis of the bacteriologic findings in 400 consecutive cases of acute nondiphtheritic laryngeal obstruction occurring in the Willard Parker Hospital between 1931 and 1934, Neffson and Wishik [1] found that there were five times as many infections due to Str. haemolyticus as to Staph. aureus. However, it was noted that in the large majority of cases of measles complicated by laryngeal obstruction Staph. aureus

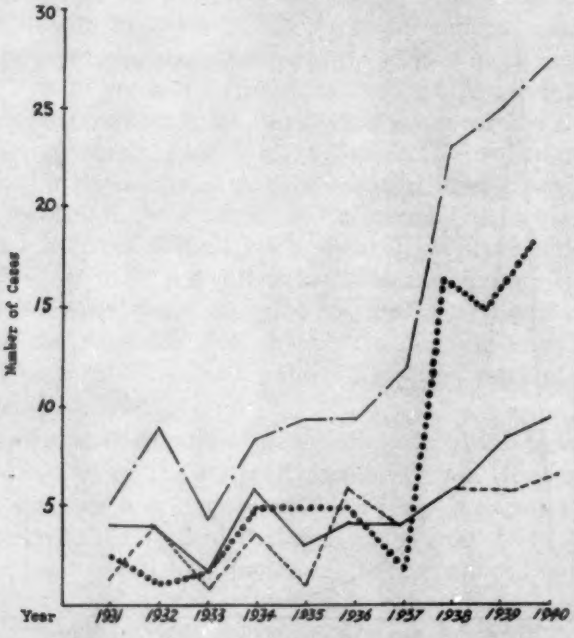

Fig. 2—Incidence of bacterial infections in cases of tracheotomy during the past decade (1931-1940). In this figure, cases of tracheotomy are indicated by a line of dots and dashes; cases in which Staph. aureus was present, by a line of heavy dots; cases in which Str. haemolyticus was present, by a solid line, and cases in which Str. viridans was present, by a line of dashes.

was present at the same time that a large preponderance of Str. haemolyticus was evident in the nonspecific cases. Moreover, it is of interest to note that the tendency to ulceration of the larynx and trachea, with or without intubation, which occurs in measles was also more prominent during the epidemics of the last three years. The possibility that Staph. aureus was a secondary invader in a primary virus infection must, therefore, be considered.

Sinclair [5] reported 10 cases in which Haemophilus influenzae type B was found in cultures of the blood taken from patients who were desperately ill with laryngitis. Special interest attaches to the fact that 8 of these cases occurred between Jan. 1, 1940 and April 1, 1941, whereas the other 2 cases were the only ones encountered during the preceding seven years.

RATIONALE OF THERAPY

An attempt was made to tide over every patient by palliative measures, including as complete rest as possible, use of a cleansing enema, mild sedation, administration of plenty of fluids, vigorous antipyretic therapy, breathing of warm moist air or oxygen, serum therapy and chemotherapy. When this treatment was not sufficient and there was enough stenosis to require mechanical intervention, three procedures were used, in the following order: aspiration, intubation and tracheotomy. Until the possibilities of aspiration were exhausted, intubation was not performed, and tracheotomy was resorted to only when aspiration and intubation failed to give sufficient relief or were deemed inadvisable. The only indication for intubation is subglottic edema. When mucopurulent secretion is present, treatment with combined suction and intubation is used.

Tracheotomy was performed as an emergency measure for relief of extreme supraglottic edema in cases in which an intubation tube was not only valueless but dangerous.[8] Tracheotomy after intubation was performed by choice under the following conditions: (1) when there was repeated blocking of the intubation tube by mucopurulent secretion, with danger of asphyxia; (2) when there was too frequent coughing up of the intubation tube, with danger of asphyxia; (3) when supraglottic edema increased enough to overlap the top of the intubation tube; (4) when there was beginning laryngeal ulceration; (5) when there was danger of a child's becoming a chronic tube carrier; (6) when the use of the intubation tube gave insufficient relief, for some unknown reason, and (7) when the child reacted alarmingly to the first asphyxial bouts.

Orton and associates,[6] citing Negus,[7] stated:

> Every patient with sufficient laryngeal or tracheal involvement to cause audible stridor on exertion is considered in the danger zone. If the stridor is noticeable

5. Sinclair, S. E.: Haemophilus Influenzae Type B in Acute Laryngitis with Bacteremia, J. A. M. A. **117**:170 (July 19) 1941.

6. Orton, H. B.; Smith, E. L.; Bell, H. O., and Ford, R. A.: Acute Laryngotracheobronchitis, Arch. Otolaryng. **33**:926 (June) 1941.

7. Negus, V. E.: An Explanation of the Respiratory Failure Sometimes Occurring After a Successful Tracheotomy, Ann. Otol., Rhin. & Laryng. **47**:608 (Sept.) 1938.

when he is resting, tracheotomy should be considered. If it is audible when he is asleep, the operation is urgently required.

If my associates and I had held this criterion for tracheotomy, the series would have included not 126 but much nearer 1,000 cases. Many a night we have sat up with a child with acute laryngeal obstruction, and when we heard a loud stridor and the patient slept, we felt relieved. It indicated that there was still a good margin of safety. It was when the stridor became "muffled," aphonic, that we began to worry, because it meant that the airway was becoming narrowed by edema or secretion or membrane or by a combination of them.

Our indications for operative intervention were as follows:[1]

Signs of extreme obstruction
- Restlessness
- Cyanosis—pallor
- Deep retractions
- Absence of breath sounds
- Anxious facies

Signs of exhaustion
- Tachycardia; impalpable pulse; heart failure
- Tachypnea—rapid, shallow breathing
- Hyperpyrexia—from 105 to 107 F.
- Stupor; coma; convulsions

Each child shows signs of anoxemia in its own peculiar manner —usually the same each time. Therefore, the signs of obstruction in any case must be evaluated individually in order to determine the most suitable time for intervention.

REASONS FOR TRACHEOTOMY

Of the 126 cases in which tracheotomy had been performed, intubation had previously been carried out in 93 and had not been done in 33 (table 2). The reasons for tracheotomy are listed as follows:

A. Cases in Which Intubation Had Not Been Done.—1. Extreme supraglottic edema on admission, requiring urgent tracheotomy: 30 cases (24 per cent of the series), with survival in 15 and death in 15; mortality rate, 50 per cent.

2. Profuse mucopurulent secretion for which intubation was deemed ineffective: 1 case, in which the patient lived.

3. Subglottic edema occurring with an acute infection of the upper respiratory tract two months after a previous tracheotomy: 1 case, with recovery.

4. Moribund state; cyanosis; larynx not examined; urgent tracheotomy: 1 case, with death.

B. Cases in Which Intubation Had Previously Been Done.—1. Extreme supraglottic edema, which developed or increased after intubation: 15 cases, with survival in 12 and death in 3; mortality rate 20 per cent. The duration of intubation was from one to eight days.

2. Too frequent coughing up of the tube, with danger of sudden asphyxia: 24 cases, with survival in 20 and death in 4; mortality rate 17 per cent. In most cases there was also a moderate amount of mucopurulent secretion; in a few the secretion was abundant. The number of intubations (before tracheotomy) ranged from 2 to 19, with an average of 9 intubations. The duration was from two to fifteen days, with an average of eight days. One patient died of asphyxia during the night, eight months after operation. The tracheotomy tube was observed to be completely blocked with mucopus. Neither the number of intubations nor the duration of intubation had any demonstrable effect on the mortality.

3. Too frequent blockage of the intubation tube by mucopus, with danger of asphyxia: 25 cases, with survival in 10 and death in 15; mortality rate 60 per cent. The number of intubations (before tracheotomy) varied from 1 to 12, with an average of 3 intubations. The duration of intubation was from a few hours to eight days, with an average of one and one-half days. Thus, the indication for operation appeared much earlier in this group than in the group in which there was too frequent coughing up of the tube—an average of one and one-half days as compared with eight days. It is interesting to note that neither the number of intubations nor the duration of intubation had any demonstrable effect on the mortality.

4. Beginning laryngeal ulceration: 12 cases, with survival in 11 and death in 1; mortality rate 8.5 per cent. The duration of intubation varied from two to eighteen days, with an average of seven days. It should be noted that of the 214 cases in which intubation was employed during the past decade (121 cases in which intubation but not tracheotomy was done, plus 93 cases in which intubation and later tracheotomy were performed), tracheotomy was required because of beginning laryngeal ulceration in only 12 (6 per cent). This ulceration was seen most frequently on the arytenoid cartilages and the aryepiglottic folds, less often on the false cords and least often on the true cords.

5. Extreme subglottic edema for relief of which it was felt that intubation would be too protracted, the danger of sudden asphyxiation following coughing up of the tube was too great and tracheotomy would be required eventually: 7 cases, with survival in 6 and death

in 1; mortality rate 14 per cent. The number of intubations varied from 1 to 3; the duration of intubation was from three hours to nine days, with an average of four days.

6. No relief from intubation: 2 cases, in both of which the patient died. Postmortem examinations were not made.

7. Repeated intubation over a prolonged period, with danger of chronicity: 2 cases, with survival in 1 and death in 1. One child had 20 intubations over a period of sixteen days and was in excellent condition before tracheotomy; bilateral pneumothorax developed immediately after tracheotomy, and the child died. The other child had 4 intubations over a period of sixteen days and made an uneventful recovery.

8. Deep cyanosis immediately after extubation (either autoextubation by coughing or trial extubation by the operator): 6 cases, with survival in 5 and death in 1; mortality rate 17 per cent. The number of intubations varied from 1 to 4, with an average of 3; the duration of intubation was from two to three days.

Résumé.—The highest mortality rate (60 per cent) occurred in those cases in which intubation was performed and tracheotomy was then required because of repeated blockage of the intubation tube by profuse mucopurulent secretion. An interesting point was that neither the number of intubations nor the duration of intubation seemed to have any prognostic bearing. Just as many patients died when tracheotomy was done early, after 1 or 2 intubations, as when the tracheotomy was done after 7 to 10 intubations, over a period of six to eight days. This does not necessarily imply that an earlier tracheotomy in some of these cases would not have been life saving. However, the answer seems to lie in the fact that cases of this type are the most serious with which one has to deal. In most cases there is a moderate amount of secretion, and in a number of cases the intubation tube becomes blocked two or three times and then the secretion diminishes. In cases of this type, however, there is usually severe laryngotracheobronchitis, with profuse outpouring of mucopurulent secretion, which blocks the entire respiratory tract, down to the smallest bronchi. With this there are often widespread bronchopneumonia and septicemia, and any type of mechanical therapy at one's disposal at present is likely to result in failure. However, tracheotomy is the treatment of choice in cases of this type.

The next most frequently fatal type was that in which such extreme supraglottic edema existed that intubation was deemed futile and an immediate tracheotomy was performed. Of 30 patients, 15 died—a mortality rate of 50 per cent. In comparison, of 15 patients who were intubated for a period of one to eight days and then had increasing

supraglottic edema, necessitating tracheotomy, only 3 died, a mortality rate of 20 per cent.

Cases in which tracheotomy was performed because of too frequent coughing up of the intubation tube, cyanosis following intubation, extreme subglottic edema, deemed to require eventual tracheotomy, and beginning laryngeal ulceration carried a mortality rate of 17, 17, 14 and 8.5 per cent, respectively.

LARYNGEAL ULCERATION

The statement has often been made that intubation leads to laryngeal ulceration. That this is a factor cannot be denied, even though the incidence of ulceration following intubation was only 6 per cent in this series. However, it should be understood that ulceration also occurs without intubation—as a result of the virulence of the infection—and can be a factor in delaying tracheal decannulation. Figures 3 to 6 are presented to show ulceration of the larynx, trachea and tracheal bifurcation in nonintubated patients with acute obstructive laryngotracheitis due to nonspecific infections, such as measles, scarlet fever and chickenpox.

Of the 10 patients for whom more than three months was required before decannulation could be performed, only 1 was tracheotomized because of laryngeal ulceration. The ulceration was noted eight days after intubation, and the tracheotomy tube was removed after nine weeks. However, a few days later pneumonia and laryngotracheobronchitis with severe subglottic edema developed. The patient was reintubated for four days and then tracheotomized again because of repeated coughing up of the tube (six times). After that she could not be decannulated for a whole year because of subglottic stenosis, which persisted in spite of laryngeal dilation. When seen recently—six years after her illness—the child was very hoarse and the trachea was extremely indrawn. Laryngoscopic examination was not permitted, but roentgenograms showed no obvious laryngeal stenosis. In 2 other patients scarring of the false cords was seen several months after tracheotomy. One had been intubated five times over a period of nine days and the other eight times over a period of five days. The voices were good. Decannulation could not be performed for one year because of narrowing of the trachea just above the tracheotomy wound, accompanied in 1 case by a subglottic web and in the other by tracheal granulations.

Thus, of the 11 surviving patients (of a total of 12) who were tracheotomized because of beginning laryngeal ulceration, only 1 required cannulation for a long period (one year); in 2 other patients, tracheotomized for reasons other than ulceration and requiring cannulation for one year, scarring of the false cords was noted several months after tracheotomy.

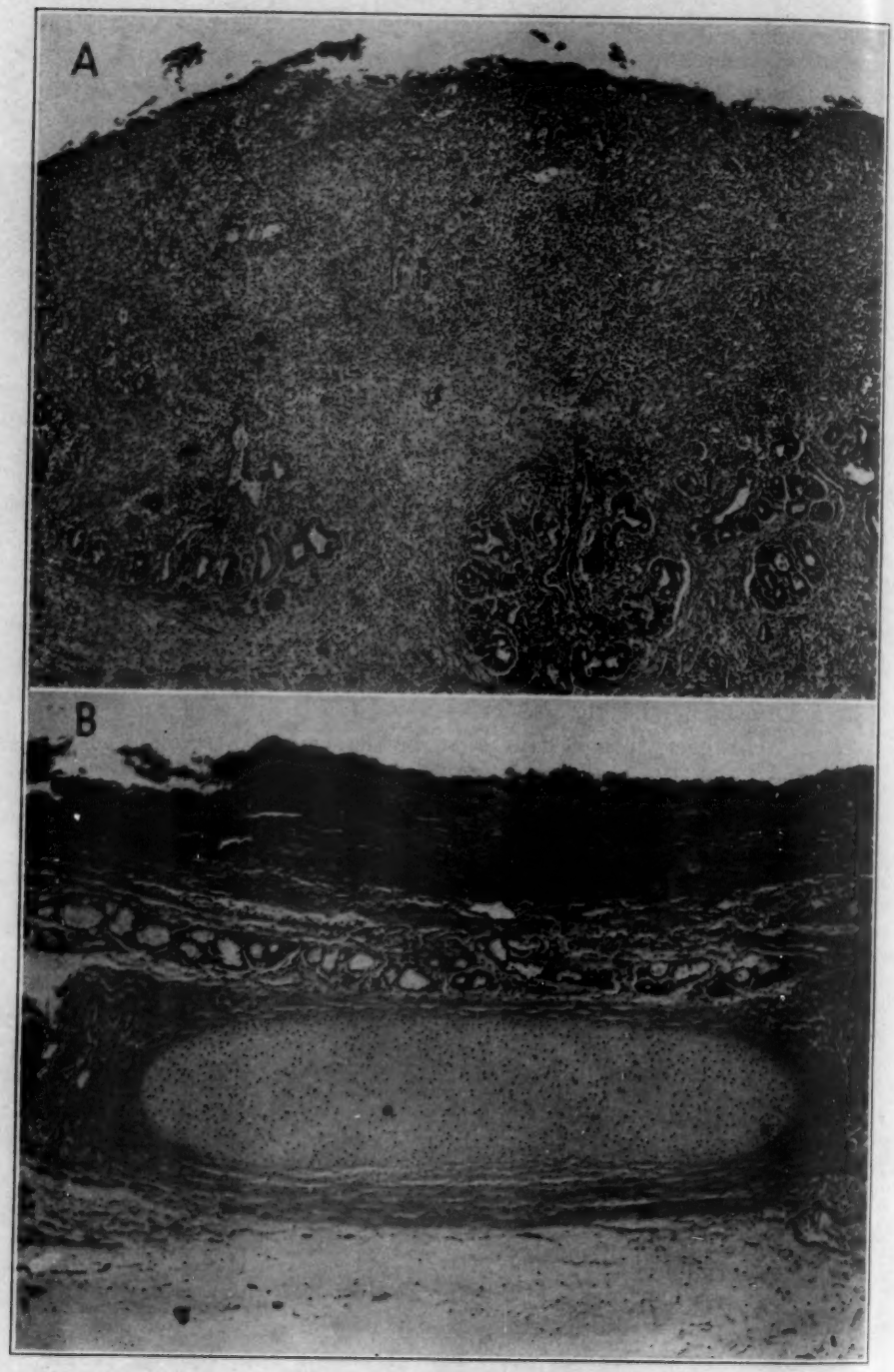

Figure 3

(See legend on opposite page)

DECANNULATION

Duration of Tracheal Cannulation.—For the 17 nonintubated patients who survived, the duration of cannulation was from six to forty-nine days, with an average of fifteen days.

For 55 (85 per cent) of the 65 intubated patients who survived, the duration of cannulation was three months or less. The period of intubation was from three hours to eighteen days, with an average of four and seven-tenths days. The period of cannulation was from seven to ninety-one days, with an average of twenty-four and five-tenths days.

For 10 patients (plus 1 patient who died eight months after tracheotomy), the duration was over three months. The period of intubation was two to sixteen days, with an average of seven and six-tenths days. The period of cannulation was six to twenty-six months, with an average of eight months.

Reasons for Difficulty in Decannulation.—Of the 11 cases in which decannulation could not be performed for three months or longer, the cause was narrowing of the trachea, usually accompanied by prevertebral edema, in 5 cases (figs. 7 to 13). This tracheal stenosis was complicated by a subglottic web in 2 cases, a subglottic stricture in 1 case, subglottic and tracheal granulations in 1 case and a subglottic web and tracheal granulations in 1 case. Of the remaining 6 cases, the cause was subglottic stricture in 3 instances, subglottic infiltration in 2 instances and tracheal granulations in 1 instance. (A more complete description is given in the section on "Difficulties in Decannulation," page 806).

CHOICE OF INTUBATION OR TRACHEOTOMY OR BOTH

During the past few years any discussion of the treatment of acute obstructive laryngotracheobronchitis has invariably led to a difference of opinion as to the choice between intubation and tracheotomy. In the earlier part of the past decade the tendency was to intubate rather than to perform tracheotomy, but during the last few years the pendulum appears to have swung in the opposite direction. As a rule, the pediatricians urged intubation and the laryngologists tracheotomy. I believe it would be valuable to examine in some detail the advantages and dis-

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EXPLANATION OF FIGURE 3

A, section of larynx in a case of acute streptococcic laryngotracheitis, showing metaplasia and ulceration of the epithelium and infiltration of the submucosa, mucous glands and muscles with plasma cells.

B, section of trachea in a case of acute obstructive (streptococcic) laryngotracheobronchitis, showing necrosis of epithelium and membrane formation.

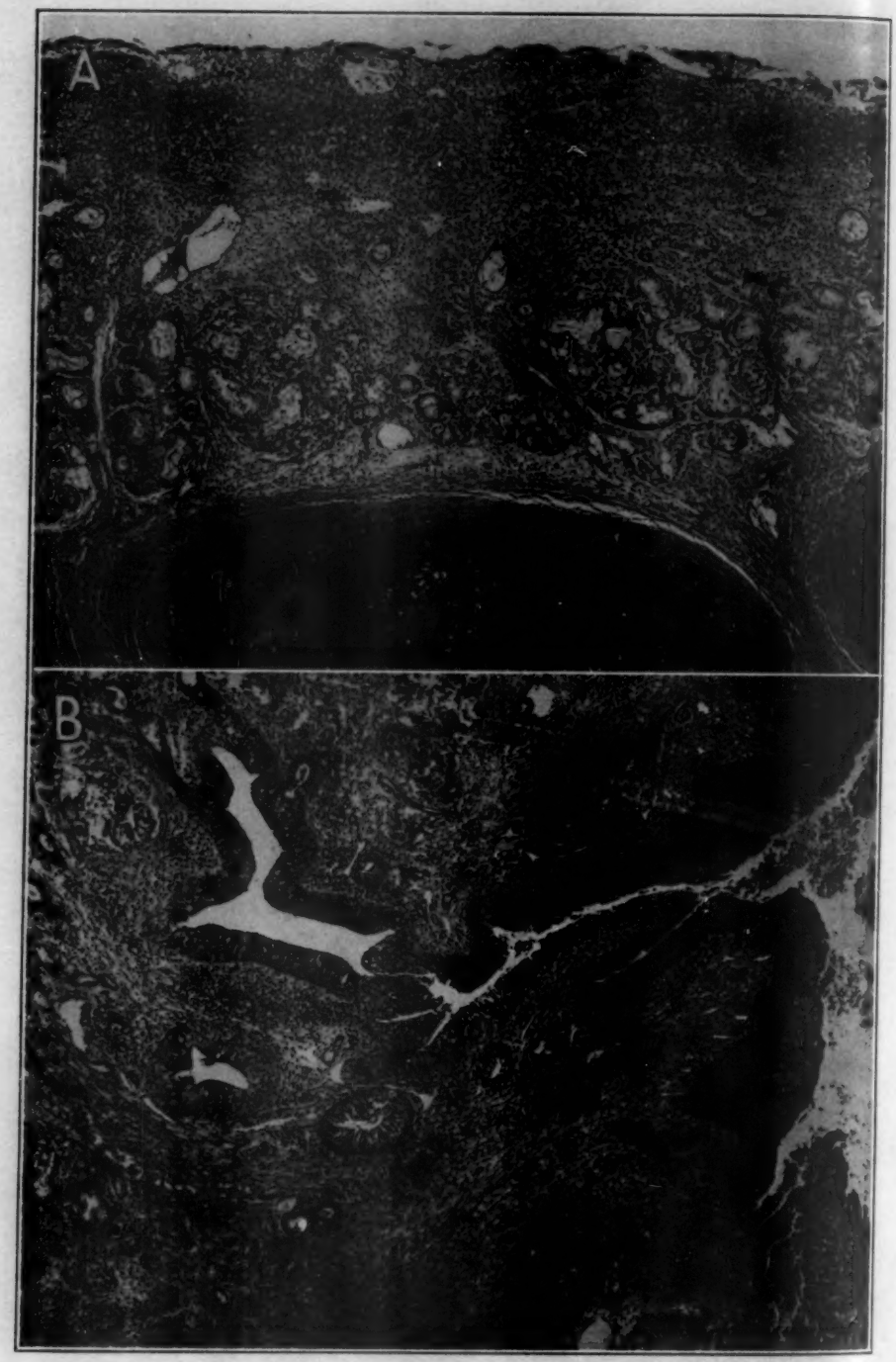

Figure 4

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advantages that each procedure possesses. Thus, perhaps, a comprehensive conception of the indications for the choice of each method can be gained.

Intubation.—Indications: First, intubation should be used only when the obstruction is due to subglottic edema. This occurs in about 85 to 90 per cent of all cases in which surgical intervention is required. When the obstruction is supraglottic intubation is extremely dangerous and usually ineffective.[8] Second, when intubation is used it must be combined with laryngotracheal aspiration; otherwise the secretions may block the tube and lead to asphyxia. (Several workers condemning intubation did not employ laryngotracheal aspiration and, naturally, had poor results.) Moreover, if these secretions are abundant, with danger of frequent blockage, tracheotomy should be done. Last, intubation should be used only when specially trained residents or attending physicians are immediately available at all times should there arise an urgent need for reintubation after the tube is coughed up, for extubation and laryngotracheal aspiration after blockage of the tube by secretions or for tracheotomy because of supervening supraglottic swelling. Unless these three conditions obtain, intubation is definitely contraindicated, except as a temporary measure, and tracheotomy is the method of choice.

Disadvantages: It being assumed that the ideal conditions just outlined prevail, intubation has certain drawbacks and definite advantages as compared with tracheotomy. The disadvantage most frequently emphasized is the occurrence of laryngeal ulceration and chronic laryngeal stenosis, preventing tracheal decannulation after tracheotomy. Other dangers not so often mentioned are sudden asphyxia from coughing up of the tube or blocking of the tube by secretion, and aspiration pneumonia due to the inability of the larynx to close during deglutition when the intubation tube is in situ. The latter danger is almost entirely eliminated by instituting gavage during the period of intubation. The former dangers can be met if expert attention is immediately available at all times.

In a review of my experience with 214 cases in which intubation was employed during the past ten years, laryngeal ulceration was found in

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EXPLANATION OF FIGURE 4

A, section of the tracheal bifurcation in a case of acute obstructive laryngotracheobronchitis, showing diffuse cellular infiltration with monocytes and polymorphonuclear cells and fibrinopurulent exudate replacing the epithelium and the necrotic duct of a mucous gland.

B, section of the larynx in a case of measles with ulcerative laryngotracheobronchitis and abscess of the lung, showing diffuse infiltration with plasma cells, denudation of epithelium, infiltration and destruction of mucous glands, with desquamation of the glandular epithelium.

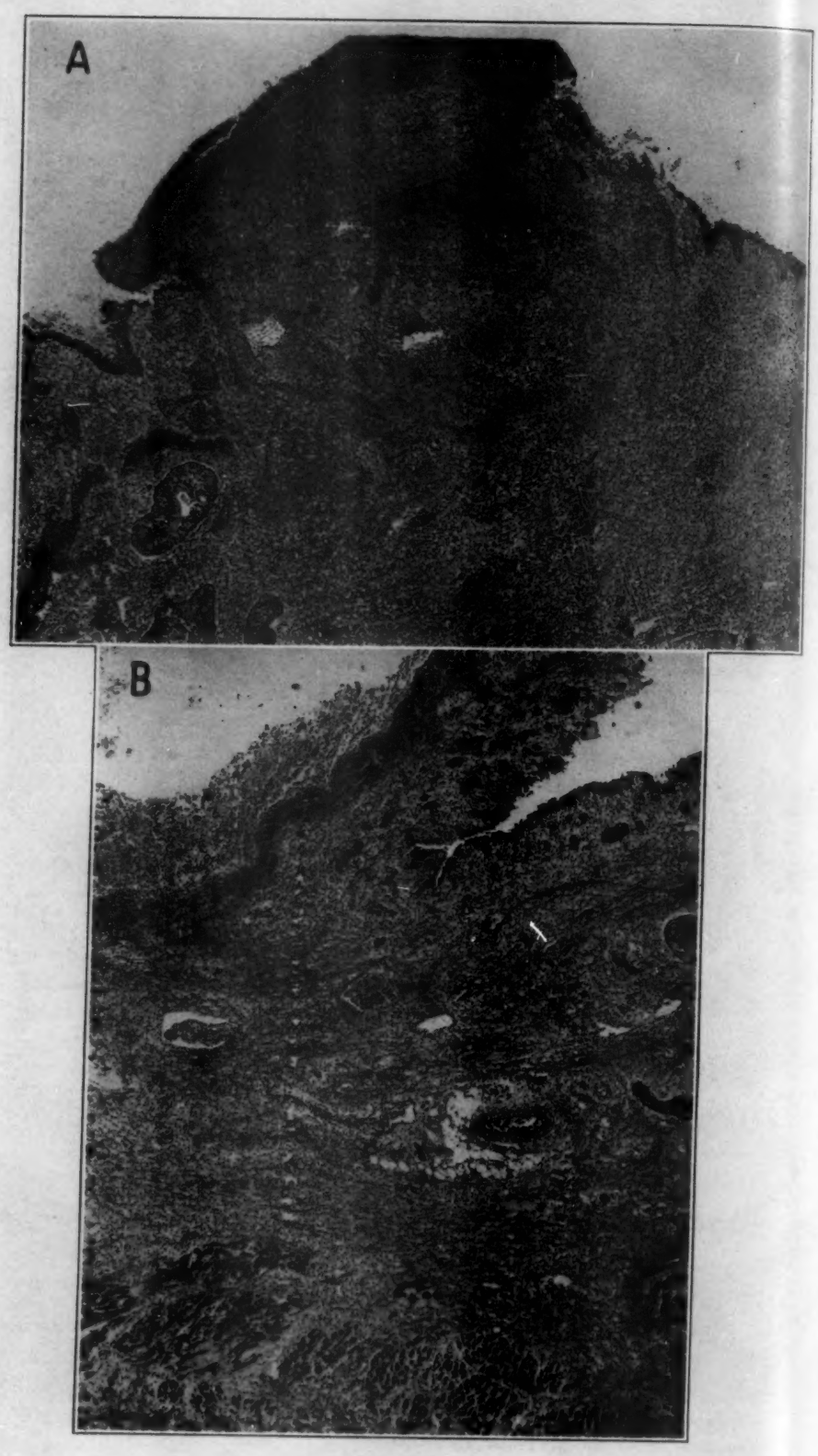

Figure 5

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only 12 instances (6 per cent), and in these cases tracheotomy was done as soon as the ulceration was discovered (in two to eighteen days, or an average of seven days, after intubation). In only 1 of these 12 cases was prolonged tracheal cannulation (one year) required, after a series of unusual incidents, already described. It might be mentioned in passing that I have had very young children who had been intubated as many as fifteen times and over a period of fifteen days without the development of either ulceration or stenosis. A great deal depends on the skill and judgment of the operator.

To estimate fairly the incidence of chronic laryngeal stenosis, one should determine the number of cases among the entire surviving group of intubated patients, whether tracheotomized or not, in which stenosis developed. In general, duration of tracheal cannulation is probably the best measure of laryngeal stenosis. Of 71 cases in which intubation was employed (over an average period of six days) plus 65 cases in which first intubation was tried (over an average period of five days) and later tracheotomy was performed, in 10 tracheal cannulation was required for more than three months. On the assumption that the prolonged tracheal cannulation was due solely to laryngeal stenosis caused by the intubation, the incidence would be only 8 per cent. However, as already noted, there were other reasons for the delayed decannulation, such as marked prevertebral swelling, causing tracheal compression, and tracheal granulations and scarring, due to the irritation of the tracheotomy tube. Moreover, it should be remembered that ulceration occurs in the larynx and trachea in cases in which intubation is not done, as a result of the infection itself, as shown in figures 3 to 6. Furthermore, who has not seen delayed decannulation in cases of very young children after a low tracheotomy without previous intubation? Truly, the occurrence of chronic laryngeal stenosis has been unduly emphasized as a deterrent to the use of intubation. A word of caution is necessary. In the 17 cases in which intubation was not done and the patient survived tracheotomy, the duration of cannulation was from six to forty-nine days, with an average of fifteen days. These cases were all of the supraglottic type of edema and cannot be compared with the cases in which intubation was done, which were all of the subglottic type. In edema of the supraglottic type the swelling is due to "waterlogging" of the tissues, which subsides

EXPLANATION OF FIGURE 5

A, section of larynx in a case of scarlatinal laryngotracheobronchitis, showing denudation of epithelium, generalized edema and diffuse polymorphonuclear infiltration extending down to the perichondrium, together with abscess formation.

B, section of larynx in a case of varicella with acute obstructive laryngotracheobronchitis and severe supraglottic edema, showing infiltration, necrosis and denudation of epithelium, monocytic infiltration between muscle bundles, deposition of fibrin and purulent phlebitis.

rather readily, whereas in the subglottic type there is usually cellular infiltration in addition to serous exudation, which requires a longer time for resorption.

Advantages: The advantages of intubation are obvious. It is simple, can be done quickly, is not much of a tax on the distressed patient, causes no surgical shock and leaves no scar. In the 71 cases in which intubation was done and the patient survived, the average duration of intubation was six days. With the advent of chemotherapy a reduction in this period of morbidity should occur.

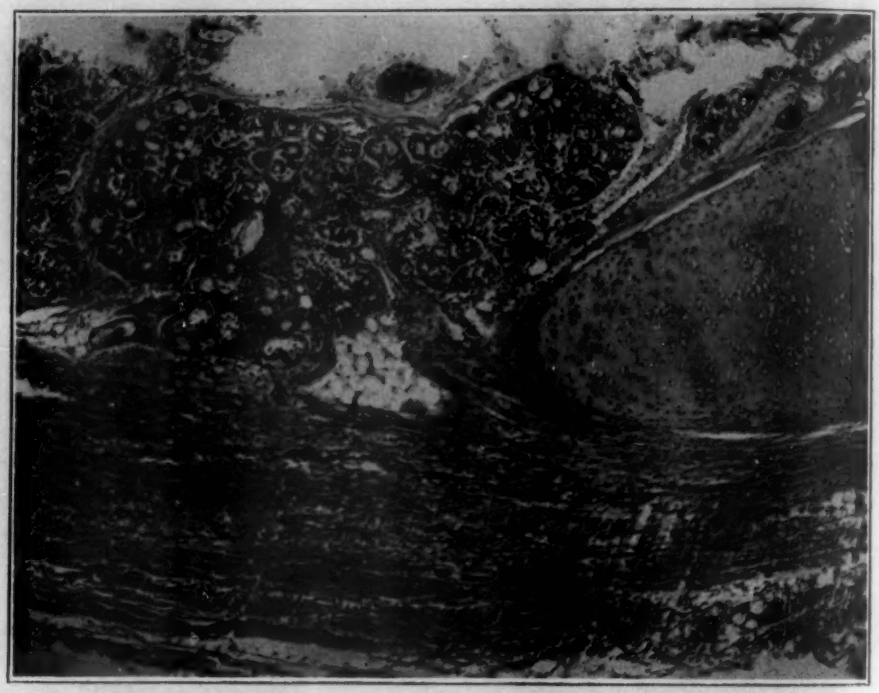

Fig. 6.—Section of trachea in the same case as that in figure 5 *B*, showing necrosis and denudation of epithelium, generalized edema and infiltration and desquamation of the glandular epithelium.

Laryngologists tend to perform tracheotomy rather than intubation because of lack of familiarity with intubation and its problems and the desire to feel comparatively free from worry about sudden asphyxia. Moreover, after a patient has died I have heard the remark, "Well, even a tracheotomy couldn't save him," as though a tracheotomy was the greatest gift that medical therapeutics could bestow on any patient. As a matter of fact, I have seen cases in which an avoidable tracheotomy followed by poor postoperative care led to a fatal ending.

Tracheotomy.—Tracheotomy has its advantages and its disadvantages. The main advantages are the ease with which thick tracheobronchial secretions can be removed and the comparative safety from sudden asphyxia, so that the patient requires less constant expert attention. (As previously noted, when there is profuse outpouring of tracheobronchial secretions tracheotomy is the procedure of choice. However, these advantages are offset by the fact that after tracheotomy there is always a great increase in the tracheobronchial secretions, requiring more frequent removal, and there is the constant danger of drying up or gumming of the secretions, resulting in plugging of the smaller bronchi.

Fig. 7.—Roentgenogram, showing marked subglottic edema.

Moreover, sudden fatal blocking of the tracheotomy tube by thick mucopus can occur, particularly at night, or the tube can slip out or be pulled out by young children or irrational adults, with fatal results. (These experiences are actual, not imaginary.) Furthermore, tracheotomy carries with it a certain amount of surgical shock, particularly in children in acute respiratory distress; there is the danger of fatal tension pneumothorax, of unsightly postoperative scarring in females and of delayed decannulation due to tracheal granulations and tracheal stricture from irritation of the tracheotomy tube.)

Statistical Analysis.—A review of the statistical aspects of this question reveals the following facts: If the mortality rate for tracheotomy in cases in which previous intubation was done is compared with that

for cases in which intubation was not done, a difference of 18.5 per cent in favor of the former (30 per cent, as against 48.5 per cent; table 2) is found. However, the cases are not entirely comparable, since in the

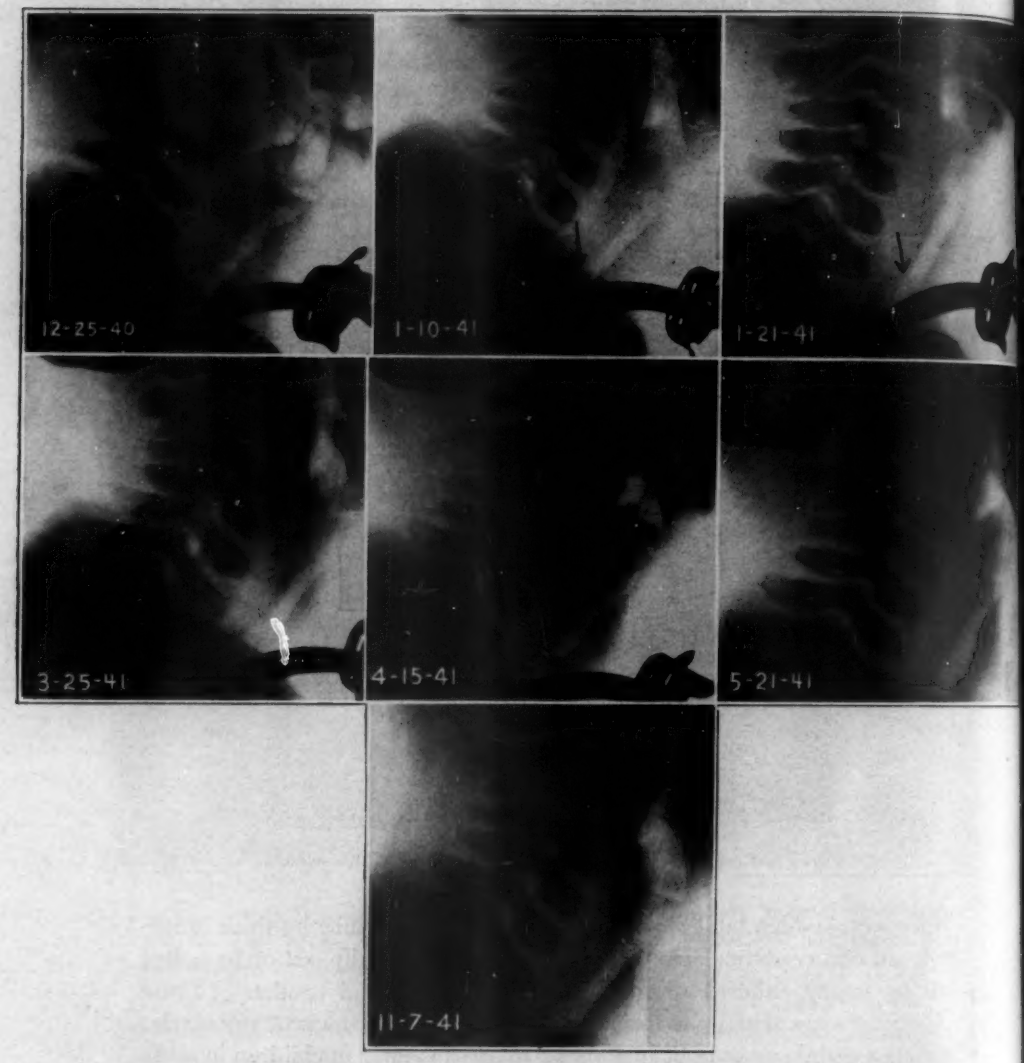

Fig. 8.—Roentgenograms, showing prevertebral edema of the neck, compression of the cervical portion of the trachea and granulation tissue around the tracheotomy opening.

cases in which intubation was not done extreme supraglottic edema was present and in the cases in which intubation was done subglottic edema occurred, at least in the beginning. In 15 cases in which intubation

was done and supraglottic edema requiring tracheotomy later developed, the mortality rate was 20 per cent; in 30 cases of severe supraglottic edema requiring tracheotomy on admission or soon after, the mortality rate was 50 per cent. But here, too, the cases are not completely comparable. The mortality rate for tracheotomy at the Willard Parker Hospital for the first seven years and that at the Kingston Avenue Hospital during the same period were 50 and 41 per cent respectively (table 4). During the last three years, 1938 to 1940, when intubation fell into disfavor at the Kingston Avenue Hospital (the frequency decreasing

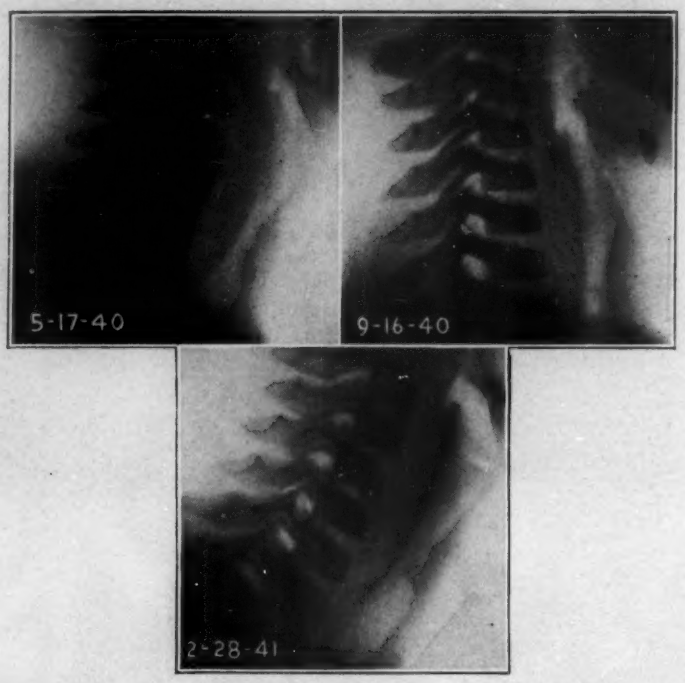

Fig. 9.—Roentgenograms, showing tracheal compression by prevertebral edema.

from 16 to 4.5 per cent, while it remained practically unchanged at the Willard Parker Hospital), the mortality rate for tracheotomy rose to 57 per cent at the Kingston Avenue Hospital and fell to 24 per cent at the Willard Parker Hospital. During this period (1938 to 1940) the frequency of tracheotomy tripled at the Kingston Avenue Hospital and quadrupled at the Willard Parker Hospital.

From these statistics one would be inclined to conclude that intubation performed before tracheotomy—in selected cases—is beneficial in that it gives the organism a chance to fight the acute infection before it is compelled to cope with the added burden of an operative procedure

and to accustom itself to an alien respiratory mechanism and physiology, with all that this implies.

Orton and associates [6] have recently come to the same conclusion, namely, that a combination of intubation and tracheotomy is the procedure of choice. However, they expressed the opinion that tracheotomy should be performed within a few hours in every case in which intubation is done. Having seen so many excellent results with intubation alone, I feel that if the obstruction is due mainly to subglottic edema a trial

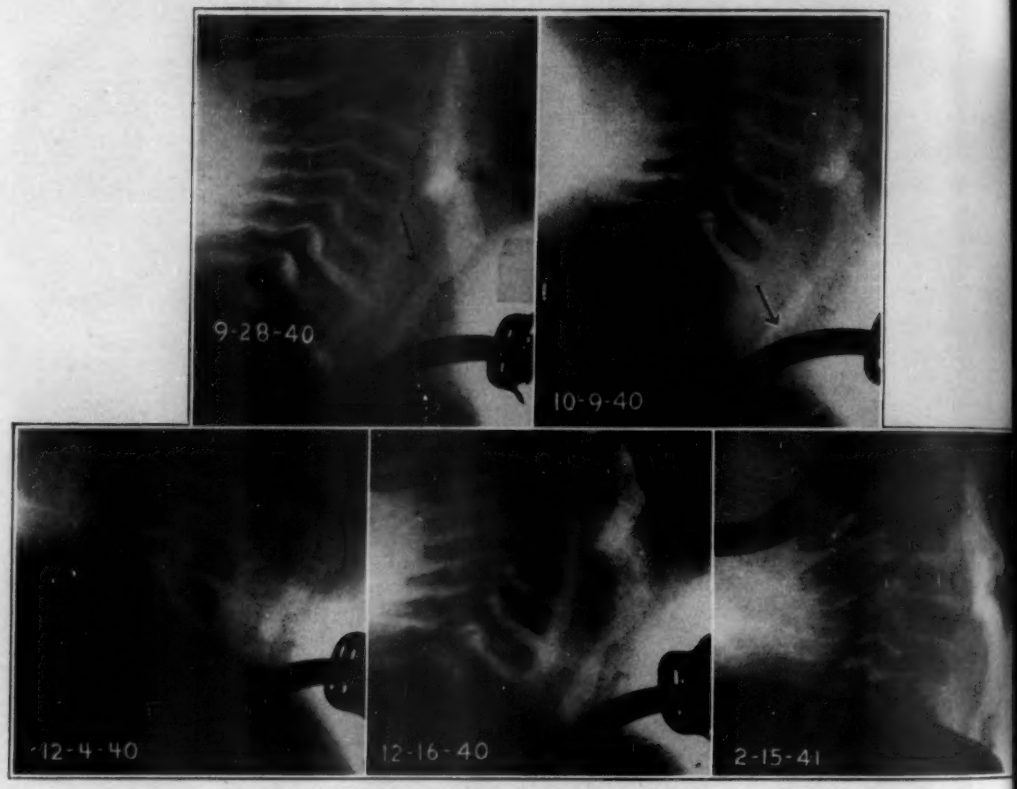

Fig. 10.—Roentgenograms, showing a subglottic web, prevertebral edema with narrowing of the trachea and tracheal granulations.

period of at least four to five days can safely be given, although in some cases I have had good results even after an intubation period of fifteen days.

After carefully weighing the advantages and dangers connected with intubation and with tracheotomy and considering a rather extensive intimate experience in the care of patients with acute obstructions of the respiratory tract, I feel that if the ideal conditions previously described can be met, intubation should be tried before resorting to tracheotomy.

MANAGEMENT OF TRACHEOTOMY

Preoperative Care.—In addition to the usual routine treatment for acute laryngeal obstruction, as outlined by Neffson and Wishik,[1] Brennemann and associates [4] and others, the patient should have, if possible, a routine roentgenographic examination to determine the presence of pneumonia, atelectasis, pneumothorax and mediastinal or pulmonary emphysema. A mild sedative should be given, but morphine and atropine are contraindicated. The temperature should be taken, so that if it is

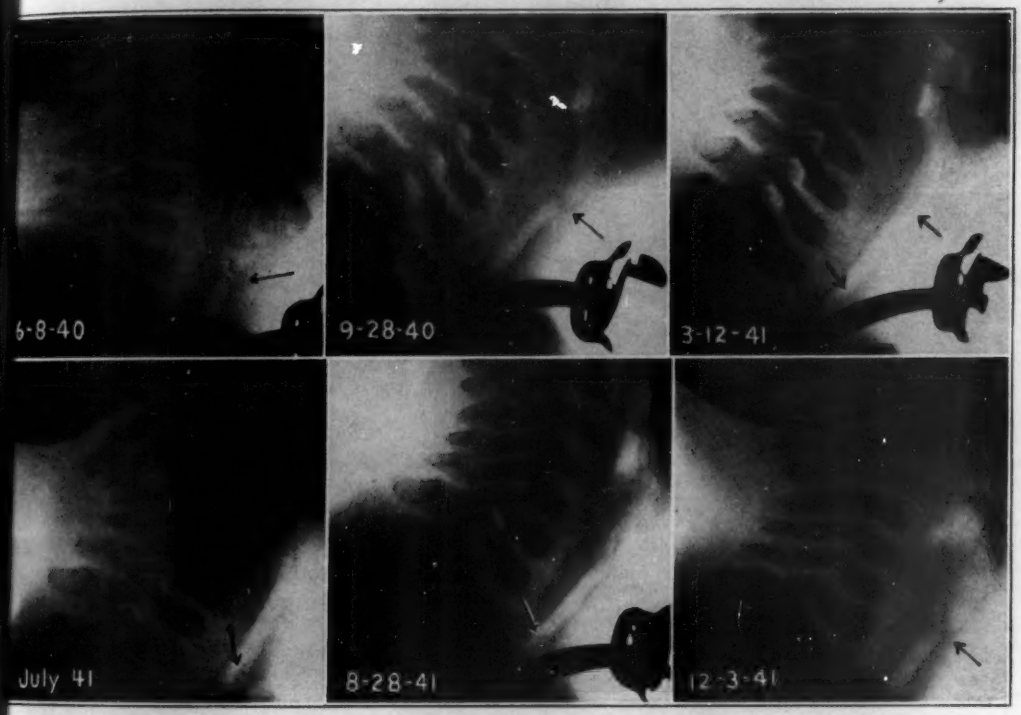

Fig. 11.—Roentgenograms, showing a subglottic stricture, prevertebral edema with tracheal compression and granulation tissue around the tracheotomy opening.

found to be high a dangerous postoperative rise may be warded off by proper antipyretic measures.

Operation.—Knowledge of the straightforward procedure of tracheotomy is taken for granted. Some suggestions usually not made follow.

1. A metal tube, such as a bronchoscope or a Willard Parker laryngeal suction tube, should be passed into the trachea to insure an adequate airway and permit an unhurried tracheotomy.

2. Local anesthesia should be used.

I recall the case of a 10 month old infant with a mediastinal abscess which was compressing the trachea and causing severe respiratory obstruction. The

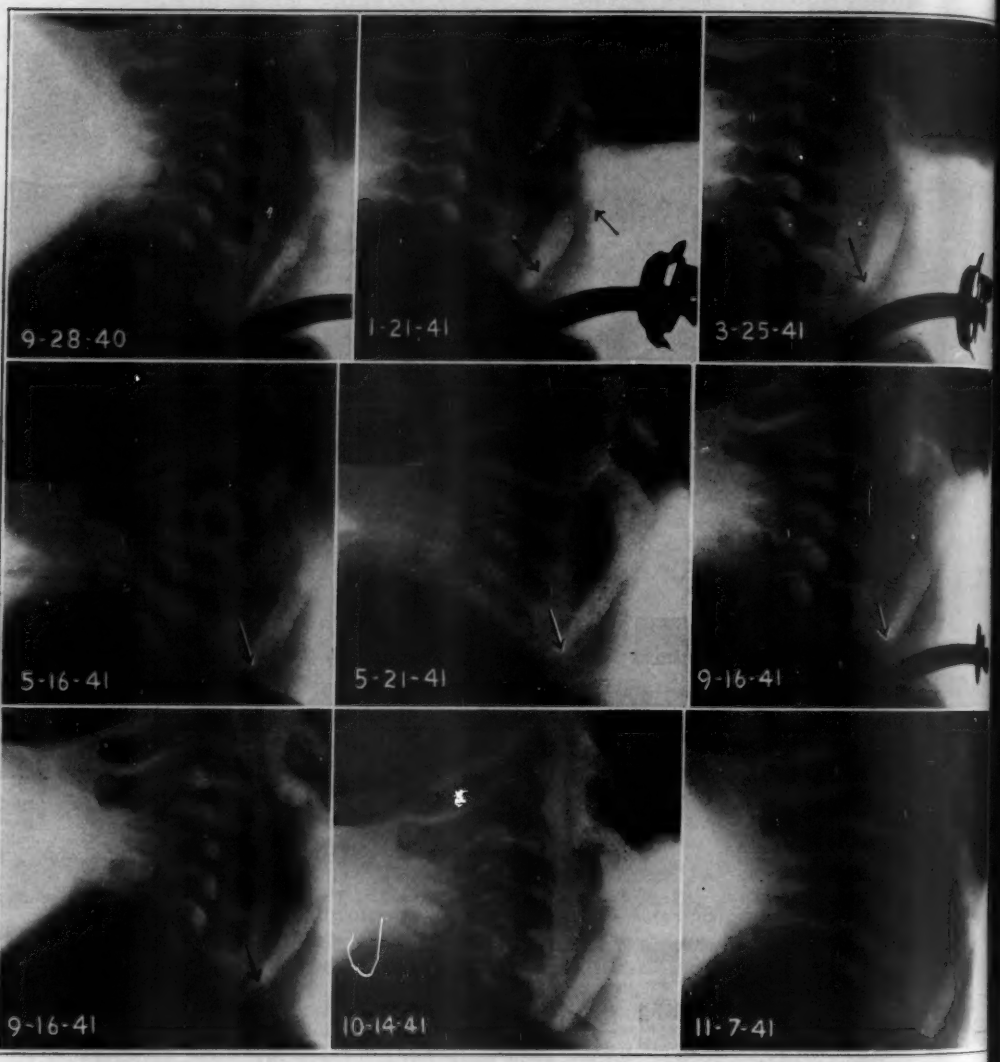

Fig. 12.—Roentgenograms, showing a subglottic web, compression of the trachea by prevertebral edema and granulation tissue around the tracheotomy opening.

surgeon ordered the anesthetist to administer ether. She tried three times and each time had to stop because the patient stopped breathing. I happened to be present and suggested that I be permitted to insert my Willard Parker laryngeal

suction tube (infant size) and that local anesthesia be used. This was done, and the operation proceeded smoothly and successfully.

3. An anatomic dissection, with the demonstration and spreading wide of each distinct fascial layer, particularly the pretracheal, should be avoided. Such dissection is extremely dangerous in that it predisposes to the production of mediastinal emphysema and pneumothorax by opening a pathway for air to be sucked down. Instead of the pretracheal fascia being separated, this fascia and the tracheal rings should

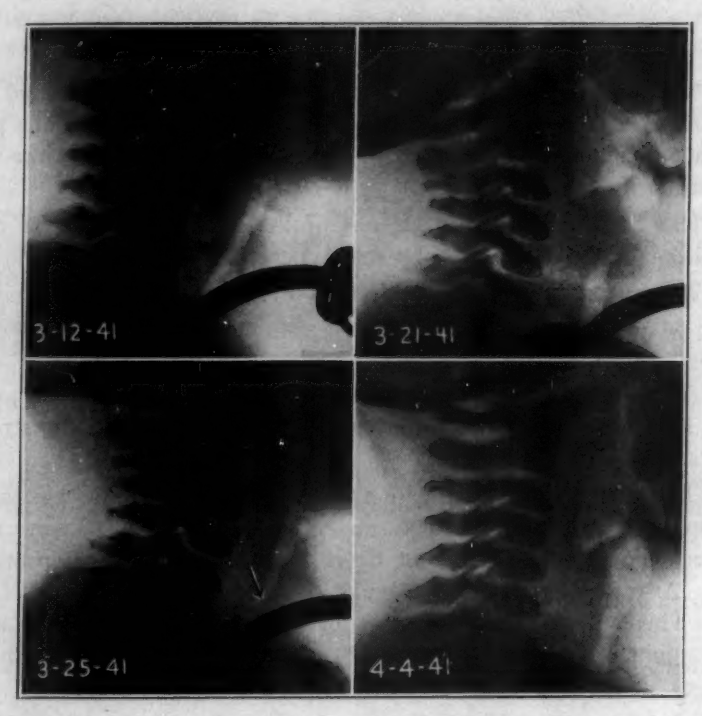

Fig. 13.—Roentgenograms, showing prevertebral edema with tracheal compression and granulations around the tracheotomy tube.

be incised at the same time. This should be done from below upward in order to avoid the possibility of nicking the pleura.

4. Removal of a circular piece of tracheal cartilage instead of cutting through the tracheal rings seems to have definite advantages, as described by Waldapfel.[8] He stated:

[With the usual vertical incision of the trachea] the C shaped cartilaginous rings, which are open at the back and connected there only by the membranous wall of the trachea, are dislocated by the cannula. The pressure causes the soft

8. Waldapfel, R.: The Opening of the Anterior Tracheal Wall in Tracheotomy, Surg., Gynec. & Obst. **71**:191 (Aug.) 1940.

membranous wall of the trachea to bulge forward toward the lumen of the trachea thus making it narrower. The cut edges of the elastic cartilaginous rings in front are bent by the pressure of the tube and the frequent change of tube may result in a narrowing of the lumen. The cut edge of the split cartilaginous rings on which the tube is continuously rubbing undergoes inflammatory changes and becomes covered with granulations. The cartilage around the tube becomes soft, is easily destroyed, and later is replaced by connective and scar tissue. [The scar tissue] may contribute to the formation of stenosis. Even if no stenosis results, the anterior wall of the trachea may lose its cartilaginous support and, after removal of the tube, move in and out with each inspiration and expiration. [These conditions become definite and serious if the tube has to be worn for a long time. . . . The excision makes changing the tube easier and it is entirely harmless.]

5. It is advisable to pack iodoform gauze into the wound around the tracheotomy tube for forty-eight hours in order to help seal off the pretracheal fascial space and prevent air from being sucked into the mediastinum.

6. Use of sutures or skin clips is optional but should not be considered dangerous, as asserted by some operators.

7. The custom of instilling cocaine into the trachea just before incising is unwise because the cough reflex is diminished and blood may be aspirated.

8. After the tracheotomy tube is inserted, it should be tested to prove that it is in the trachea by noting that the blast of air is full and strong, like a strong puff.

9. The tape should be tied with a triple knot to prevent any possibility of slipping and should fit rather snugly around the neck. I have seen the tracheotomy tube pulled out by children, and once by a maniacal adult. Also, the tie should be made in the center of the back of the neck. This will remove the chance of twisting the tracheotomy tube, which might occur if the knot is tied on the side of the neck, so that it impinges on the wall of the trachea.

10. Persistent coughing following the insertion of the tube may be due to the fact that the tube is resting on the carina. In that case, several layers of gauze, placed between the flange of the tube and the skin, can be used to "build up" the tracheotomy tube.

Warning: One must be sure the inner tube is exactly as long as the outer tube. If it is the least bit shorter, dried secretion will accumulate and block the distal end of the outer tracheotomy tube. When the nurse removes and cleans the inner tube, the outer tube will remain blocked, even after she inserts a soft rubber catheter into the trachea. I have seen such occurrences end almost fatally.

Postoperative Care.—Special nurses, intelligent and specially trained, should be on duty constantly. This is essential for good results. They should be given careful, minute instructions in every case and be told to

call the physician at once if any perplexing situation arises. The nurses should be instructed to place the tips of the fingers over the tracheotomy tube to test the volume and force of the air blast, which should always be like a strong puff. If it is diminished, and is not improved materially by suction, the nurse must inform the physician at once. A bronchoscopic set should be at hand in case of emergency.

A. Routine Care: 1. Medical treatment: (*a*) General measures. (1) Rest must be encouraged as much as possible. The sleeping child should not be disturbed unless absolutely essential. To aid rest, administration of mild sedatives, such as bromides or phenobarbital, is advisable. Morphine, codeine and atropine should not be given. They depress the valuable cough reflex, diminish the manifestation of restlessness as an index of air hunger and result in drying and gumming of secretions.

(2) Feeding is preferably oral—by bottle, Breck feeder or medicine dropper with a rubber catheter tip, as well as by nasal or oral gavage if necessary. In older children it may also be rectal. Parenteral administration may be by hypodermoclysis or, better, by intravenous injection, which is less disturbing. Care must be taken not to give too much fluid parenterally, for fear of overburdening the heart. If the tracheotomy has followed intubation, nasal gavage should be used for the first day or two. This is done to remove the danger of aspiration, for the larynx is sometimes unable to close properly for a day or so after intubation.

(3) Oxygen, if necessary, can be supplied by catheter or funnel into the tracheotomy tube. The oxygen should be allowed to bubble through warm water before reaching the trachea. Holinger and associates[9] asserted that oxygen increases the viscosity of the tracheobronchial secretions and that carbon dioxide diminishes secretion by stimulating its resorption, liquefies the sputum and stimulates the cough reflex. They maintained that when oxygen is used, both steam and carbon dioxide are necessary to neutralize the drying effect of the oxygen, neither one being sufficient by itself. A concentration of 5 to 10 per cent of carbon dioxide is used.

(4) Transfusions, if indicated, should be small and repeated at frequent intervals as needed.

(5) Antipyretic measures may be called for. In 16 of the cases (13 per cent) the temperature rose to from 105 to 108 F. after tracheotomy. This complication must be watched for by taking temperatures routinely immediately after operation and hourly thereafter for the first four to six hours. Vigorous antipyretic measures should be started at

9. Holinger, P.; Basch, F. P., and Poncher, H. G.: The Influence of Expectorants and Gases on Sputum and the Mucous Membranes of the Tracheobronchial Tree, J. A. M. A. **117**:675 (Aug. 30) 1941.

once. In cases in which the temperature is over 104 F. acetylsalicylic acid, wet packs, ice caps, or even ice cold enemas, should be used, depending on the case.

(*b*) Specific measures. (1) Convalescent scarlet fever serum seems to have some value in infections due to Str. haemolyticus.

(2) Staph. aureus antigen has been used in selected cases, with occasional striking results.

(3) Sulfonamide compounds, i. e., sulfanilamide, sulfapyridine (2-[paraaminobenzenesulfonamido]-pyridine), sulfathiazole (2-[paraaminobenzenesulfonamido]-thiazole) and sulfadiazine (2-[paraaminobenzenesulfonamido]-pyrimidine), have been used at the Willard Parker Hospital since 1937. Of this series, 81 cases in which tracheotomy was performed have been observed since the start of sulfonamide therapy. Chemotherapy was not used in 28 cases, in 11 of which the patient died, a mortality rate of 40 per cent; it was given in inadequate doses in 11 cases, in 1 of which death occurred, a mortality rate of 9 per cent, and it was given in adequate doses in 42 cases, in 9 of which death occurred, a mortality rate of 21 per cent. From these statistics it is difficult to draw conclusions as to the effect of the drug on the mortality rate. Many patients died before they could be given the drug. Inadequate therapy was due to toxic manifestations, lack of improvement or acute aggravation of the illness. However, in the 11 cases in which treatment was inadequate the mortality rate was only 9 per cent. (Of course, this is too small a series from which to draw any conclusions.) Since it has been shown that in the past few years there has been a decided increase in the incidence of Staph. aureus infections, the use of sulfathiazole, and recently of sulfadiazine, has been favored. At the Willard Parker Hospital the drug was used in adequate concentration for a period of three to seven days and was not required after that time. As soon as toxic symptoms occur it should be stopped. Also, if the patient's condition is not better, or perhaps is even worse, after chemotherapy has been used with adequate concentrations for twenty-four to thirty-six hours, it is wiser to discontinue the treatment, for it will probably be of no benefit. Orton and associates[6] expressed the belief that " . . . although sulfanilamide and sulfapyridine are not specifics for this disease, they seem to have some value." At the Willard Parker Hospital my colleagues and I feel that chemotherapy has resulted in a decrease in complications, particularly pneumonia and septicemia, and has probably lowered the mortality rate. However, the local condition has not been affected sufficiently to lessen the need for mechanical intervention.

2. Mechanical intervention: All mechanical treatment is directed toward keeping the airway completely clear at all times. To do this,

secretions must be removed and drying prevented. This can be accomplished by the following means:

(*a*) The foot of the bed should be elevated about 20 to 25 degrees, as tolerated by the patient, and lowered, if all is well, after four to six days.

(*b*) The patient should be in a warm, moisture-laden atmosphere, with the temperature about 70 to 75 F. and the relative humidity 80 to 90 per cent. If the room is too warm, it is depressing both to the patient and to the nurses. An ordinary room humidifier can be combined with a steam vaporizer to obtain the proper warmth and humidity. If the patient is given oxygen, carbon dioxide and moisture should be added, as previously noted.

(*c*) The tracheotomy tube should be covered with several layers of loose gauze or a sea sponge and kept moist constantly with warm saline solution.

(*d*) Several drops of warm 5 per cent solution of sodium bicarbonate is dropped into the tracheotomy tube on inspiration, every fifteen to thirty minutes the first day and then less often, as necessary. This usually causes coughing and moistens the tracheobronchial secretions. After the instillation, secretions in the trachea are aspirated with a soft rubber catheter. The catheter should be pinched off during the insertion (to prevent it from sticking to the wall of the trachea) and should be pushed down, during inspiration, into the bronchi, first on one side and then on the other, by tilting the tracheotomy tube or turning the patient. Before inserting the catheter it is a good idea to place the finger over the tracheotomy tube during the coughing spell initiated by the solution. This will give the child something to cough against and make the cough more effective. If gumming and crusting of the secretions have occurred, limited lavage of the tracheobronchial passages can be tried. From 5 to 15 cc. of the 5 per cent solution of sodium bicarbonate should be instilled during inspiration, with the head elevated, and then aspirated, with the head lowered. The instillation of sodium sulfathiazole has been recommended and is worthy of trial in staphylococcic infections. In rare cases the main bronchi may become so edematous as to prevent aspiration even by the smallest bronchoscopic suction tubes available. In these cases several drops of a solution of epinephrine hydrochloride (1:5,000 to 1:1,000) should be instilled regularly every half hour or so, as needed.

(*e*) The inner tracheotomy tube should be removed, examined and cleaned every half-hour for the first two days and then less often, as necessary.

(*f*) Gummed or hardened secretions which cannot be aspirated by a rubber catheter must be removed by a metal bronchoscopic tube or for-

ceps. This condition must be thought of when the air blast becomes weak and cannot be improved by the usual tracheal aspiration. (Pneumothorax is another possibility.)

3. Roentgenographic examination: Routine roentgenograms of the chest should be taken as soon after tracheotomy as possible and as often thereafter as warranted. In this way earlier diagnosis of pneumothorax, mediastinal emphysema and massive atelectasis can be made and proper treatment begun at once.

B. Unusual Complications—Warnings: 1. Acidosis and alkalosis: In patients with prolonged respiratory obstruction acidosis tends to develop as a result of the accumulation of carbon dioxide and of acids formed because of overwork and the diminished intake of bases and fluids. Particularly in very young children, the acid-base balance is easily upset and must be watched carefully. A much less common occurrence, but more serious, because it is often overlooked, is the development of alkalosis. In several cases, after tracheotomy for prolonged respiratory obstruction the patient continued to breathe rapidly and deeply for long periods. This hyperventilation, with the blowing off of large amounts of carbon dioxide, resulted in alkalosis, accompanied by the usual signs of tetany, such as carpopedal spasm, twitchings and even convulsions. Such a patient should be given a carbon dioxide–oxygen mixture to breathe, saline solution by mouth or intravenously and calcium gluconate. Saline solution is preferred to lactate–Ringer's solution (Hartmann's solution) because the latter leaves undesired base after being metabolized. The carbon dioxide concentration and the carbon dioxide–combining power of the blood should be followed carefully. Of course, this treatment should be under the guidance of a pediatrician or an internist.

2. Tension pneumothorax and mediastinal emphysema: The treatment of these complications has been described elsewhere.[2]

3. Use of complete tracheotomy tube: As is well known, the tracheotomy tube consists of an inner and an outer tube, in order to facilitate cleaning. About a year ago I was called in consultation to see a 5 month old infant who was having extreme respiratory distress after tracheotomy. On examination I found that the otolaryngologist had inserted only the inner part of a no. 1 tracheotomy tube, because, he said, "the outer tube was too large." On removal, this tube was found to be completely blocked with hardened secretion. A complete no. 1 tracheotomy tube was inserted, and the respiratory distress was entirely relieved. A few months later I had an almost identical experience. A 10 month old infant, a physician's child, was in extreme respiratory distress. The otolaryngologist was found to be using only the outer part of a no. 1 tracheotomy tube. He stated that when he

inserted the inner tube the child could not breathe at all. On removal of this tube, it, too, was found to be blocked with hardened secretion. A complete no. 2 tracheotomy tube was inserted and "built up" with several layers of gauze. The infant was immediately relieved and made an uneventful recovery. Thus, the complete tracheotomy tube must be used at all times.

4. Apnea: After prolonged apnea, the patient may not regain consciousness. This is probably due to minute cerebral hemorrhages. Thorner and Lewy [10] stated:

> Exposures to sublethal periods of pure anoxia lead to vascular and degenerative changes in the brains of guinea pigs and cats. Some of these changes are irreversible and become summated.

Similar changes probably occur in human beings. If the respiratory center is involved, there may be shallow, irregular respirations. In such a case the use of a mechanical respirator is indicated.

5. Restlessness and convulsions: Extreme restlessness and convulsions are serious complications. They may be due to cerebral hemorrhages following prolonged anoxia or alkalosis, hyperpyrexia, air hunger, beginning meningitis, tension pneumotherapy or chemotherapy. These possibilities must be borne in mind and appropriate measures taken. In this series, 9 of the patients manifested extreme restlessness after tracheotomy; 4 of these also had generalized convulsions. Three of the 4 children with generalized convulsions died. Of the 3 who died, 1 had convulsions in conjunction with bilateral tension pneumothorax; another had convulsions before and after tracheotomy, of unknown origin; the third had an overwhelming amount of mucopurulent secretion due to acute laryngotracheobronchitis. One patient with extreme restlessness had influenzal meningitis; another, high fever, tetanic spasms and twitchings nine days after tracheotomy; in a third the extreme restlessness and thrashing about lasted twenty-four hours and was thought to be due to sulfathiazole. It disappeared on discontinuance of the drug.

6. Accumulation of secretions in trachea: After the tracheotomy tube has been removed and the wound closed, it occasionally happens that respiratory distress occurs a day or so later, even though the child has done well with the tube completely blocked for several days. This is usually due to the accumulation of tracheobronchial secretions which the patient is unable to cough up. Aspiration of the trachea and bronchi, either through a bronchoscope or by means of a Willard Parker suction tube, should be performed and repeated as often as

10. Thorner, M. W., and Lewy, F. H.: The Effects of Repeated Anoxia on the Brain, J. A. M. A. **115**:1595 (Nov. 9) 1940.

necessary. Usually, two or three times has been sufficient to insure recovery. In 1 case, that of a 6 month old infant, the tracheotomy tube was removed and the wound closed two months after the operation. One day later respiratory distress developed and the laryngotracheobronchial airway was aspirated, with complete relief. This had to be repeated twice, but the third time the aspiration was ineffectual and an emergency tracheotomy had to be performed by the resident. However, the child died. Postmortem examination revealed thick mucopus in the trachea and bronchi, which was the cause of the asphyxiation. There was also some granulation tissue around the tracheotomy wound, but no tracheal stenosis.

C. Decannulation: 1. Routine procedure: After the temperature curve is flat for three days and the child appears to be doing well, the tracheotomy tube is gradually blocked with some adhesive tape. This should be done while the patient is asleep or is distracted. In this way the psychic factor is eliminated, and any difficulty in breathing through the larynx can be noted. If all is well, a direct laryngoscopic examination is done to make sure there are no abnormalities, such as swelling, ulceration, web or stricture. In most cases the tube can be completely blocked at once, and after several hours it can be removed and the tracheotomy wound closed. In some cases the child has to be "weaned." This is done by covering more and more of the tracheotomy opening by means of adhesive tape. A grooved rubber stopper can also be used, but I have found the adhesive tape simple and satisfactory. If the partial blocking does not solve the problem, the use of progressively smaller tracheotomy tubes may be the answer.

2. Difficulties in decannulation: It should be mentioned here that in none of the cases was a "high" tracheotomy done.

(*a*) Fear—spasm. Nearly all children become panic-stricken when the tracheotomy tube is removed and they realize it is not going to be replaced. If such a situation is encountered the tube should be gradually blocked while the child is asleep. Then, if all is well, it should be gently removed. When the child awakes he does not realize at first that he is no longer breathing through the tube and finds later that he can breathe perfectly well through the normal air passages. Another method is to distract the child while one is removing the tube, so that he is not aware of the change in his manner of breathing.

(*b*) Narrowing of the trachea. (1) Prevertebral edema of the neck has been seen so often in cases of acute laryngeal obstruction (even without tracheotomy) that one has come to regard it as part of the disease rather than as a complication. In almost all cases it disappears with subsidence of the infection. However, in some cases it persists

and compresses the trachea, delaying decannulation. During the last two years of this study routine serial roentgenograms were taken of the larynx and the neck. In 5 of the cases of prolonged cannulation the sole or partial cause was tracheal compression by prevertebral swelling (figs. 8 to 13). In such cases diathermy can be tried to hasten absorption of the edema.

It is interesting to note, in passing, that Grier [11] found typical enlargement of the superior mediastinal shadow on roentgenograms of the chest in 34 of 59 cases of acute laryngitis. This shadow is symmetric and "projects on both sides of the spine, and the edges are usually concave, but occasionally straight." He differentiated this from the thymus shadow, which "is almost always a bulging shadow, the borders being convex. . . . It is not uncommon for it to project to one side of the spine only." I have noted the same thing, together with the prevertebral edema.

(2) Other causes of tracheal stenosis were (*a'*) the formation of granulation tissue around the tracheotomy wound and, as explained by Waldapfel,[8] (*b'*) the bulging forward of the membranous posterior tracheal wall, due to the spreading of the incomplete tracheal rings by the tracheotomy tube, (*c'*) the bending inward of the cut edges of the cartilaginous rings and (*d'*) the necrosis of the cartilage and its replacement by scar tissue, which causes further contraction.

Tracheal granulations occurred in sufficient amounts to cause obstruction and difficulty in decannulation in 3 cases (figs. 8, 12, 13 and 14).

In an infant 4 months old who had been intubated nineteen times over a period of seven days, granulations formed around the tracheotomy wound. The child was able to breathe with the tube completely blocked but had respiratory distress when the tube was removed. This occurred either because the trachea collapsed or because the granulations fell together, with blocking of the trachea. She was finally decannulated six months later.

Another child, 2 years old, had been intubated five times over a period of nine days because of extreme subglottic edema. One month after tracheotomy roentgenograms showed prevertebral edema of the neck and the cervical portion of the trachea was compressed and pushed forward. This subsided after one month (fig. 8*A* to *G*). Three months after tracheotomy granulation tissue was seen 1 to 2 cm. below the vocal cords. The voice was clear. When sitting up the child was able to breathe with the tube completely blocked or with the tube out. However, on lying down he became distressed, so that

11. Grier, G. W.: Mediastinal Enlargement in Acute Laryngotracheobronchitis, Ann. Otol., Rhin. & Laryng. **50**:941 (Sept.) 1941.

the tube had to be replaced and left patent. Six months after operation there was a great deal of granulation tissue around the wound. He was finally decannulated one year after tracheotomy, and when seen recently the boy had a good, clear voice.

The third case is described under the heading "Subglottic Stenosis—Strictures and Webs" (case 7).

Treatment in these cases consisted of (*a'*) removing the source of irritation by the use of smaller and smaller tracheotomy tubes and, finally, of "dummy" tubes; (*b'*) blocking the tracheotomy tube to the

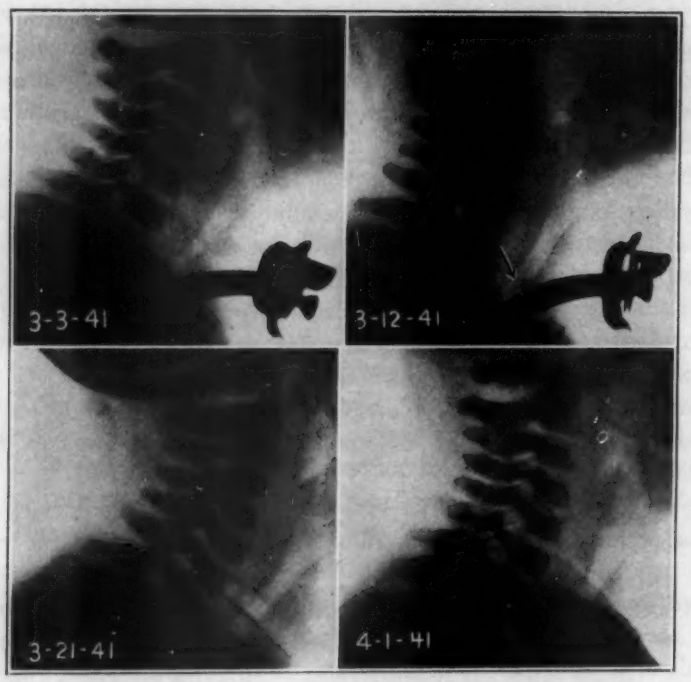

Fig. 14.—Roentgenograms, showing granulation tissue around the tracheotomy tube.

utmost tolerated by the patient to encourage the use of the larynx; (*c'*) burning the granulations with a silver nitrate stick, and (*d'*) radiotherapy to destroy and prevent reformation of the granulations.

(*c*) Subglottic stenosis. (1) Subglottic infiltration. In 3 cases the original subglottic swelling persisted for a long time, so that tracheal decannulation had to be deferred. The subglottic portion of the larynx had the appearance of two sausage-like swellings which almost met in midline (fig. 7). In the first case, that of a 4 month old Negro infant, who was intubated nine times over a period of seven days, decannu-

lation had to be postponed for five months for this reason. The larynx and voice were normal. In the second case, that of a 14 month old Negro boy, who had been intubated for sixteen days, the tracheotomy tube could not be removed for eight months because of the persistent subglottic infiltration. The larynx and voice were normal. The third case, that of a 1 year old Negro girl, is described under the heading "Strictures and Webs" (case 7).

Dilation of the larynx by bougies was tried in these cases, but it was concluded that the best treatment was to leave the larynx alone and attempt to block the tracheotomy tube as much as the child would tolerate, in order to encourage the use of the larynx and promote its growth.

(2) Subglottic granulations. This complication occurred in at least 1 case, already described in connection with tracheal granulations, and probably in several others, reported under the head "Subglottic strictures and webs."

(3) Subglottic strictures and webs. These complications occurred in 7 cases.

CASE 1.—A girl aged 1 year was intubated for eight days and then tracheotomized; decannulation was done after nine weeks. She was again intubated for four days and then tracheotomized. Subglottic stricture developed, requiring laryngeal dilation. Decannulation was done fifteen months later. On follow-up observation the child was found to be very hoarse and the trachea to be drawn in on inspiration.

CASE 2.—A girl aged 1½ years was intubated twelve times over a period of nine days and then tracheotomized. Subglottic stricture developed, requiring laryngeal bougienage at weekly intervals. Decannulation was done thirteen weeks later. On follow-up examination she was found to be moderately hoarse when excited, but the voice was strong.

CASE 3.—A child aged 1 year was intubated seven times over a period of seven days and then tracheotomized. Subglottic stricture developed, and dilation with laryngeal bougies was necessary about once a week. The child died at night, seventy days after tracheotomy. The tube was found to be blocked with thick mucopus.

CASE 4.—A child aged 3 years was intubated once over a period of three days and then tracheotomized. Subglottic stricture developed, and dilation was effected with laryngeal bougies. The trachea was compressed by prevertebral edema (fig. 9). Decannulation was done six months later. The voice was strong and clear.

CASE 5.—A child aged 2 years was intubated three times over a period of two days and then tracheotomized. A web was found 2 cm. below the vocal cords five months after tracheotomy. Dilation was effected regularly with laryngeal bougies. Prevertebral edema of the neck with narrowing of the trachea was noted nine months after tracheotomy (fig. 10). Decannulation was carried out after one year. The voice was strong and clear.

CASE 6.—A boy aged 1½ years was intubated three times over a period of three days and then tracheotomized. Six months later a subglottic stricture was noted. This was dilated at irregular intervals. One year later a thick strand of scar tissue was still present posteriorly about 1 to 2 cm. below the vocal cords, but a no. 20 F laryngeal dilator was easily passed. Nine months after tracheotomy roentgenograms showed the cervical portion of the trachea to be greatly narrowed by prevertebral edema (fig. 11). Treatment consisted in removing scar tissue with laryngeal punch forceps, application of radiotherapy, laryngeal bougienage, use of progressively smaller tracheotomy tubes and gradual blocking of the tube. At the end a small "dummy" tube was used. Decannulation was finally done twenty-two months later. His voice was strong and clear.

CASE 7.—A girl aged 1 year was intubated three times over a period of five days and then tracheotomized. Subglottic swelling was noted five months after operation. Laryngeal dilation was done at biweekly intervals, but supraglottic edema developed after each treatment and the bougienage was stopped. Nine months after operation the airway was completely blocked, but this lasted only a day or so. One year after tracheotomy the larynx was seen to be scarred, and there was a subglottic web, reducing the lumen to one-third its normal size. Her voice was clear. Laryngeal bougienage was repeated, and dilators up to no. 20 F. were passed. The subglottic stenosis diminished, but granulation tissue developed around the tracheotomy wound, causing increasing stenosis (fig. 12). One year after tracheotomy the trachea was seen to be narrowed and pushed forward by prevertebral edema (fig. 12). Radiotherapy was given but had to be discontinued because the patient was exposed to measles and varicella. The tracheotomy tube was gradually blocked to encourage use of the larynx; smaller tubes were used, and, finally, a "dummy" tube was employed. Decannulation was done twenty-six months after tracheotomy—the longest period of cannulation in any case in this series. On discharge, the voice was strong and clear.

D. Complications: The following complications were found in this series of 126 cases.

	No. of Cases
Bronchopneumonia	26
Pneumothorax	17*
Mediastinal emphysema	30*
Septicemia	12
Pleurisy; empyema	8
Mediastinitis, purulent	7
Otitis media	6
Mastoiditis	2
Myocarditis	5
Pericarditis	4
Endocarditis	2
Meningitis	3
Infarction of lung	3
Abscess of lung	2
Thymitis, acute	2

* This complication has been discussed in another article.[3]

There was 1 case of each of the following complications: abscess of the larynx, sublingual abscess, submental abscess, supraclavicular abscess, multiple abscesses, encephalitis, purulent arthritis and appendicitis.

Atelectasis was a common complication, sometimes involving an entire lobe, particularly the upper lobe of the right lung. It usually disappeared within a week, but sometimes persisted for several weeks.

MORTALITY

A. General Mortality.—1. The mortality rate for 1,360 patients with acute nondiphtheritic laryngeal obstruction was 9.5 per cent. For any full year this varied from 4.1 to 14 per cent, but, as pointed out previously, the incidence of cases of laryngeal obstruction did not have any apparent effect on the general mortality rate (table 1). Furthermore, even the number of intubations and tracheotomies could not be used as a fair index of epidemic virulence. For example, in 1937-1938, when the general mortality rate was 14 per cent, 39 per cent of the patients received either intubation or tracheotomy, 15 per cent requiring tracheotomy. In 1938-1939, the general mortality rate was only 8.5 per cent, although 36 per cent of the patients received either intubation or tracheotomy, 25 per cent requiring tracheotomy. In 1932-1933, the mortality rate was 13.5 per cent; yet only 13 per cent received intubation or tracheotomy, 3.4 per cent requiring tracheotomy. The only exception was in 1933-1934, when the mortality rate was lowest —4.1 per cent—and only 8 per cent received intubation or tracheotomy, 4.1 per cent requiring tracheotomy. A partial explanation for this lack of parallelism may be the personal equation. Some residents are more easily upset by a given amount of respiratory obstruction and therefore more apt to intubate; others are more enthusiastic about tracheotomy and tend to operate sooner.

2. The general mortality rate for tracheotomized patients was 35 per cent. The incidence of tracheotomy varied from 4.3 per cent (1931-1935) to 19.4 per cent (1938-1940). The mortality rate for tracheotomized patients during these periods varied from 56 per cent (1931-1935) to 24.3 per cent (1939-1940)—a distinct correlation. Thus, the more frequently tracheotomy was performed, the lower was the mortality rate for tracheotomized patients. However, the general mortality rate in 1931-1935 was 9.8 per cent, and that in 1938-1940, 8.5 per cent—a difference of little significance.

3. The mortality rate for tracheotomized patients previously intubated was 30 per cent, and that for nonintubated patients was 48.5 per cent (table 3).

B. Specific Factors in Mortality Rates.—The mortality rates were calculated on the basis of several factors.

1. Extent of Involvement:

	No. of Patients	No. of Deaths	Mortality Rate, Per Cent
(a) Laryngeal	9	2	11
(b) Laryngotracheal	57	6	12
(c) Laryngotracheobronchial	60	37	61.6

2. Age (years):

	No. of Patients	No. of Deaths	Mortality Rate, Per Cent
Under 1	24	12	50
1-2	43	13	30
2-3	21	7	33
3-4	16	5	31
4-10	19	6	32
10-15	1	Patient lived	..
20-30	1	1	..
47	1	1	...

Thus, under the age of 1 year the hazard from tracheotomy appears to be greatest—with a mortality rate of 50 per cent.

3. Sex:

	No. of Deaths	Mortality Rate, Per Cent
88 males	31	35
38 females	14	37

4. Temperature on Admission: No significant conclusions could be drawn as to prognosis on the basis of this factor. A few patients with temperatures of 105 to 106 F. died.

5. Period of Hospitalization Before Tracheotomy:

	No. of Patients	No. of Deaths	Mortality Rate, Per Cent
(a) Few hours	53	28	53
(b) 1 to 3 days	38	9	24
(c) 1 to 3 weeks	35	9	26

It would appear, therefore, that if tracheotomy can be postponed for a few days the outlook for life is improved.

6. Therapy with Sulfonamide Compounds (sulfanilamide, sulfapyridine, sulfathiazole): After treatment with sulfanilamide or one of

its derivatives was started, 81 cases were observed in which tracheotomy was done. The data on these cases follow:

Sulfonamide Compound	No. of Patients	No. of Deaths	Mortality Rate, Per Cent
(*a*) Not given	28	11	40
(*b*) Inadequate doses	11	1	9
(*c*) Adequate doses	42	9	21

No conclusions can be drawn from these statistics. Many patients died before they could be given any chemotherapy. Inadequate therapy was due to restlessness, lack of improvement and toxic manifestations. Of the cases in which treatment was adequate, the bacteriologic findings were as follows: Staph. aureus, 16 cases; Str. haemolyticus, 9 cases; Str. viridans, 5 cases; Pneumococcus type XIV, 1 case.

SUBSEQUENT OBSERVATIONS

Requests for the follow-up examination were sent to the parents of the 82 patients surviving tracheotomy and the 71 patients surviving intubation during the past decade. Altogether, 51 children were seen from one to ten years after their illness. Of these, 21 had been intubated, 4 tracheotomized and 26 both intubated and tracheotomized. None of the 21 intubated patients had any discernible change in the pitch or intensity of the voice; 2 gave a history of recurring attacks of mild "croup." The 4 patients who had been tracheotomized likewise showed no vocal changes. Of the 26 patients who had been intubated and later tracheotomized, 1 was moderately hoarse and another extremely hoarse, with difficulty in breathing due to retraction of the trachea. Direct laryngoscopy was not permitted, but roentgenograms revealed no laryngeal stenosis. This patient had required a second tracheotomy because of laryngotracheobronchitis and pneumonia, which developed after the first decannulation. After that she had to wear the tracheotomy tube for a whole year. In 9 of these patients there was a history of repeated attacks of mild "croup."

SUMMARY AND CONCLUSIONS

An attempt is made to give a general, comprehensive description of the various aspects of tracheotomy in cases of acute nondiphtheritic obstructive infections of the larynx, trachea and bronchi, together with detailed, practical suggestions for routine treatment and for the recognition and handling of unusual complications.

From the statistical survey presented, the following summary and significant conclusions can be drawn: Of 1,360 patients admitted to the Willard Parker Hospital during the past decade with acute non-

diphtheritic laryngeal obstruction, 126, or 9.2 per cent, required tracheotomy. The incidence of tracheotomy increased from 5.3 per cent during the first seven years to 19.4 per cent during the last three years, and the mortality rate of tracheotomy fell from 50 per cent during the first seven years to 24.3 per cent during the last three years. (During these same periods the incidence of intubation scarcely varied —8.7 to 9.4 per cent—but the mortality rate dropped from 43 per cent during the first seven years to 28 per cent during the last three years.) The fourfold increase in the incidence of tracheotomy was accompanied by lowering of the general mortality rate from 10 per cent in the first seven years to 8.5 per cent in the last three years. Probable factors in the reduction of the mortality rate are the improved postoperative care, the earlier recognition and treatment of serious complications and the use of chemotherapy.

Statistics of the Kingston Avenue Hospital during the same periods showed strikingly similar figures for the general incidence and mortality rates of acute obstructive laryngitis. Moreover, just as the incidence of tracheotomy quadrupled in the Willard Parker Hospital, it tripled at the Kingston Avenue Hospital during the last three years as compared with the first seven years. However, there were two striking differences: 1. The incidence of intubation fell from 16 per cent during the first seven years to 4.5 per cent during the last three years, whereas it hardly varied at the Willard Parker Hospital. 2. The mortality rate for tracheotomy rose from 41 to 57 per cent at the Kingston Avenue Hospital, while it fell from 50 to 24.3 per cent at the Willard Parker Hospital. It is interesting to note that the mortality rate for the tracheotomized patients who were not intubated was 48.5 per cent, while for those who were intubated it was 30 per cent (table 2).

In view of the various arguments brought forth by otolaryngologists and pediatricians and the statistics and discussions presented in this paper, it is felt that if specially trained residents are available, intubation in properly selected cases is the method of choice. By giving the organism an opportunity to fight the acute infection before compelling it to cope with the added burden of an operative procedure and an alien respiratory physiology, the chances of recovery are increased. Of course, when ideal conditions for the use of intubation do not exist, tracheotomy is to be preferred.

A remarkable increase in the incidence of infections due to Staph. aureus has been noted during the past three years. The probability that this organism is a secondary invader in a primary virus infection is discussed. The use of sulfathiazole or sulfadiazine is recommended as the treatment of choice if chemotherapy is to be used before the infecting organism has been identified.

Serious complications, often overlooked, such as extreme hyperpyrexia, restlessness, convulsions, acidosis and alkalosis, together with suggestions for their early recognition and treatment, are described. A comprehensive discussion of tension pneumothorax and mediastinal emphysema occurring after tracheotomy is given elsewhere.[2]

Laryngeal ulceration following intubation and its occurrence without intubation are discussed.

The difficulties and complications of decannulation are described, and therapy is suggested. The conclusion is reached that intubation is not the single important factor in prolonged cannulation, as is usually asserted. It is due also to (*a*) the dense infiltration of the subglottic tissues, which often occurs in such infections; (*b*) the formation of occasional strictures and webs, not due necessarily to the intubation; (*c*) the occurrence of prevertebral edema with compression of the trachea, and (*d*) tracheal stenosis around the wound because of anterior puckering of the posterior tracheal wall, granulation tissue around the wound and erosion and scarring of the cut cartilaginous rings.

The mortality rates are discussed in general and specifically, according to extent of involvement of the respiratory tract, age, sex, temperature on admission, length of hospitalization before tracheotomy and therapy with sulfonamide compounds.

Results of a follow-up study are listed. Of 153 patients who survived intubation, tracheotomy or both, 51 were seen from one to ten years after their illness. Of these, 21 had been intubated and showed no vocal changes as to quality, pitch and intensity. The 4 children who were tracheotomized likewise showed no changes. Of the 26 persons who were intubated and tracheotomized, 1 was moderately hoarse and another very hoarse; the rest showed no changes. Eleven of the 51 patients gave histories of repeated attacks of mild croup, from one to six times.

Practical suggestions for use during the performance of tracheotomy are given, and the routine postoperative care is outlined in detail.

Willard Parker Hospital.

EMBRYOLOGIC OBSERVATIONS BEARING ON THE PROBLEM OF OTOSCLEROSIS

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In the vocabulary of the otologist there are few words the meaning of which is so commonly misunderstood as that of otosclerosis. As one pages through the volumes of literature on otosclerosis one is frequently at a loss to know what the author wishes to convey by the term. So loosely is the term otosclerosis used that in one breath an author may refer to a clinical complex and in another to a histopathologic change in the otic capsule.

From a clinical point of view the term otosclerosis refers to one form of progressive hardness of hearing, in which, according to standard textbooks, the malady shows:

1. Onset in youth, of hereditary or familial character and progressive, and often bilateral, involvement.
2. Prolonged bone conduction.
3. Early loss of low tone perception.
4. Negative Rinne reaction.
5. Essentially normal condition of other structures, such as pneumatic spaces, the tympanic membrane and the middle ear, except for a reddened spot in the region of the promontory.

The term otosclerosis as used from the histopathologic point of view refers to definitely circumscribed areas of changed bone in the otic capsule. When used in this sense the true meaning of the term is expressed. Therefore, in this discussion, for the sake of distinction, the term "clinical otosclerosis" will be employed in speaking of the hard of hearing complex.

In the search for the pathology underlying progressive deafness of the type referred to as clinical otosclerosis, one pathologic condition has pretty uniformly been found, namely: The otic capsule showed one or more definitely circumscribed areas of sclerotic bone, and one of these areas invariably occurred anterior to the oval window and had spread to such an extent as to cause fixation of the stapes. Thus clinical otosclerosis has come to be associated with stapes fixation due to the pathologic condition of the bone known as otosclerosis.

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Drury,[1] in 1926, summarized the pathologic changes in so-called clinical otosclerosis. He stated that otosclerosis is a disease of the osseous capsule of the labyrinth consisting of patches of sclerotic bone. The usual site of these foci is in the wall of the labyrinth anterior to the stapes. In these areas bone is absorbed, and this is followed by deposition of new bone. In the process of absorption the cartilage lining the oval window and the foot plate of the stapes and adjacent portions of the annular ligament disappears and is replaced by bone, so that the stapes becomes fixed.

Wilson,[2] in discussing the histopathology of otosclerotic foci in the otic capsule, made this statement: "As fixation of the stapes is the chief cause of deficiency of hearing, it is this phase which has chiefly attracted the otologist."

Now it must also be pointed out that otosclerotic foci in themselves may not produce clinical symptoms. Nager[3] reported that of a large number of petrous bones studied which showed otosclerotic foci, stapes fixation occurred in only 41 per cent. He stated that only when such sclerotic foci involve the stapes ligament or impinge on the auditory nerve are clinical symptoms present.

This belief that fixation of the stapes was the chief cause of the loss of hearing induced otologists of the latter part of the past century to attempt its mobilization. The results were not uniform, and only in very early stages were they encouraging. In 1897, at the meeting of of the German Otological Society, Passow[4] reported that since mobilization of the stapes presented so many difficulties it occurred to him that the same effect might be produced by making an orifice in the labyrinthine wall. A hole was made anterior to the stapes. Results were striking at first. Auditory capacity then diminished but remained better than before operation. Passow expressed the belief that no more could have been done by mobilization of the stapes.

In 1913 Jenkins,[5] on the basis of the hypothesis that the symptoms accompanying otosclerosis are due to changes in the density of the labyrinthine fluid, treated 2 patients by opening the perilymphatic space of the external semicircular canal. The hearing was improved at first and then became worse, but remained better than before the operation.

1. Drury, D. W.: Progressive Deafness, Laryngoscope **36**:545, 1926.
2. Wilson, J. G.: Present Status of the Problem of Otosclerosis, Arch. Otolaryng. **38**:946 (Dec.) 1938.
3. Nager, F. R.: Zur Klinik und Anatomie der Otosclerose, Acta otolaryng. **27**:542, 1939.
4. Passow, in discussion on Pause, R.: Verhandl. d. deutsch. otol. Gesellsch. **6**:141, 1897.
5. Jenkins, G. J.: Otosclerosis, Tr. Internat. Cong. Med., 1913, sect. 16, pt. 2, p. 609.

Bárány in 1914, cited by Fremel [6] in 1922, had a similar experience. Holmgren [7] in 1923 opened 4 labyrinths by drilling a hole anterior to the stapes. In more recent years he opened the posterior canal. The story was the same in most cases. There was improvement at first, and then as the hole closed by growth of new bone, hearing loss occurred.

What, then, is the basis for the loss of hearing if hearing can be reestablished by merely drilling a hole into the labyrinth? Certainly, the stapes in such cases is still fixed and the ossicular chain is still impaired. Is then, the fixation of the stapes the chief responsible factor in the loss of hearing? Jenkins struck a different note when he assumed a change in the density of the labyrinthine fluid. Is it not possible that overemphasis on stapes fixation has caused otologists to overlook other possible factors?

In order to cast a different light on the problem, may I call attention to the structure of and the growth changes in the area anterior to the oval window? This is the area which has been designated as the area of predilection for the otosclerotic foci, which by their growth came to involve the stapes joint or other adjacent structures. It is one of these structures anterior to the stapes to which I want to call attention.

THE FISSULA ANTE FENESTRAM

At the angle of junction of the scala vestibuli and the vestibule the periotic tissue projects through an irregular cleft in the bony otic capsule and becomes continuous with the mucoperiosteal tissue of the middle ear just anterior to the oval window. This cleft in the otic capsule was referred to by Siebenmann [8] as the "fissula ante fenestram" (*FAF*, fig. 1 *A* and *B*). In recent years this fissula has been amply described for fetuses and young children by Bast [9] and for older children and for the adult by Anson and others [10] and Wilson.[11]

6. Fremel, F.: Demonstration eines Otosklerose von 8 Jahren operierten Patienten, Monatschr. f. Ohrenh. **56**:552, 1922.

7. Holmgren, G.: Zur Chirurgie der Otosklerose, Acta oto-laryng. **27**:338, 1939.

8. Siebenmann, F.: Die Korrosions-Anatomie des menschlichen Ohres, Wiesbaden, J. F. Bergmann, 1890.

9. Bast, T. H.: Development of the Otic Capsule: II. The Origin, Development and Significance of the Fissula Ante Fenestram and Its Relation to Otosclerotic Foci, Arch. Otolaryng. **18**:1 (July) 1933.

10. Anson, B. J., and Wilson, J. G.: The Fissula Ante Fenestram in the Adult Human Ear, Anat. Rec. **56**:383, 1933. Anson, B. J., and Martin, J., Jr.: Fissula Ante Fenestram: Its Form and Contents in Early Life, Arch. Otolaryng. **21**:303 (July) 1935.

11. Wilson, J. G.: Fissula Ante Fenestram and the Adjacent Tissue in the Human Otic Capsule, Acta oto-laryng. **22**:382, 1935.

The following facts are pertinent to the present consideration: 1. The fissula ante fenestram is a constant structure in the human otic capsule. It is specific to the human capsule. It is not present in the other animals studied, not even in the monkey. 2. The fissula is filled

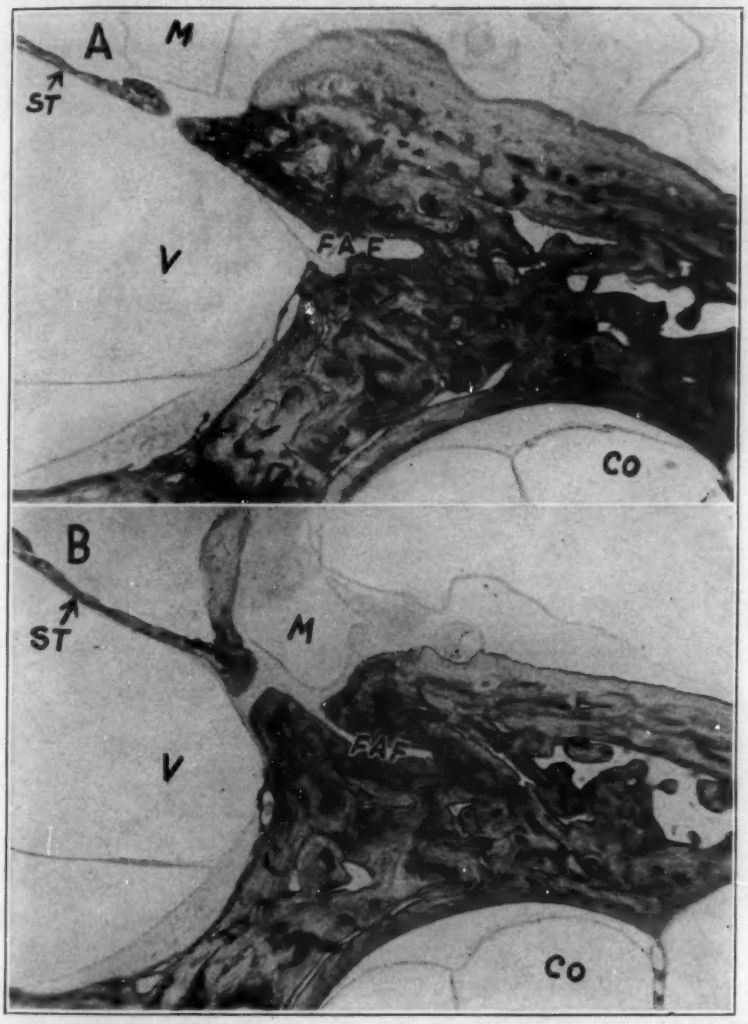

Fig. 1.—Photomicrographs of portions of histologic sections of the petrous bone of baby 93 L, aged 14 months, showing a normal fissula ante fenestram (*F A F*).

A (slide 31; section 2) shows the fissula opening to the vestibule (*V*); *B* (slide 35; section 4), the fissula opening to the middle ear (*M*).

St indicates stapes, and *Co*, cochlea.

with a loose periotic tissue at its vestibular end and a somewhat denser tissue at its tympanic end, where it becomes continuous with the muco-periosteal tissue. In the ears of fetuses and children at least, many endothelium-lined, capillary-like spaces can be seen in the fissular tissue. Whether these spaces are arterial, venous or lymphatic has not been

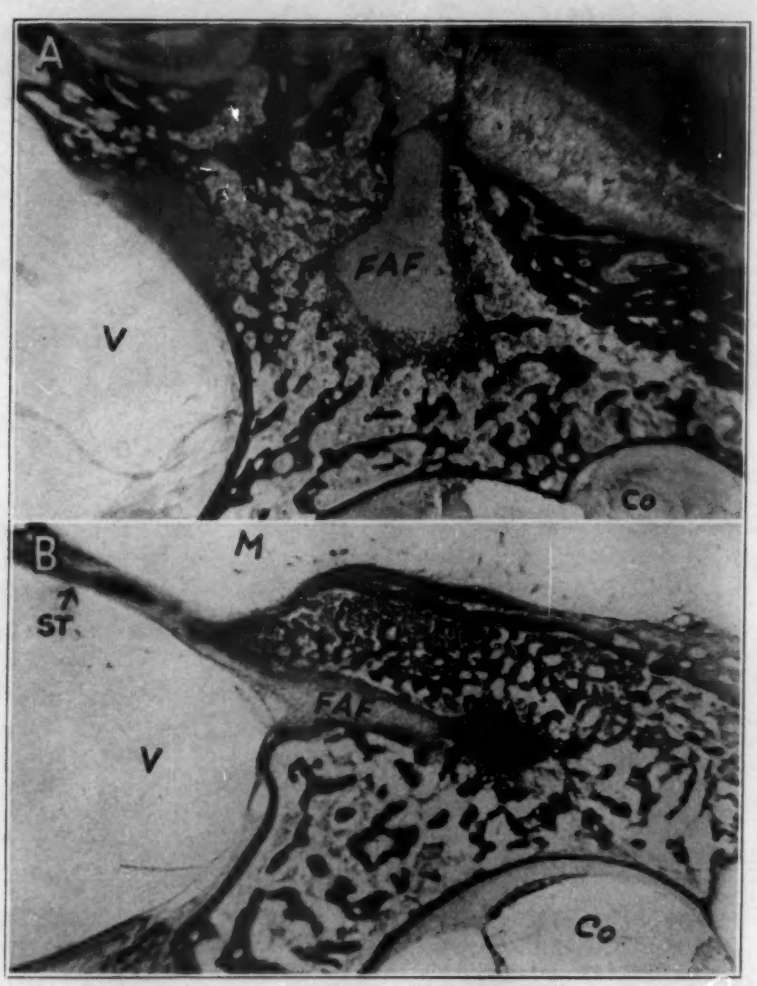

Fig. 2.—*A* (embryo 45 B, aged 29 weeks; slide 33, section 5) shows a large fissula (*F A F*) opening into the middle ear. The fissula is filled with young cartilage.

B (embryo 70, aged 23 weeks; slide 28, section 1) shows the large fissula (*F A F*) opening into the vestibule (*V*). In the cochlear end of the fissula a dark-stained mass of mature cartilage can be seen.

M indicates middle ear; *M M*, mucosa of the middle ear; *Co*, cochlea, and *St*, stapes.

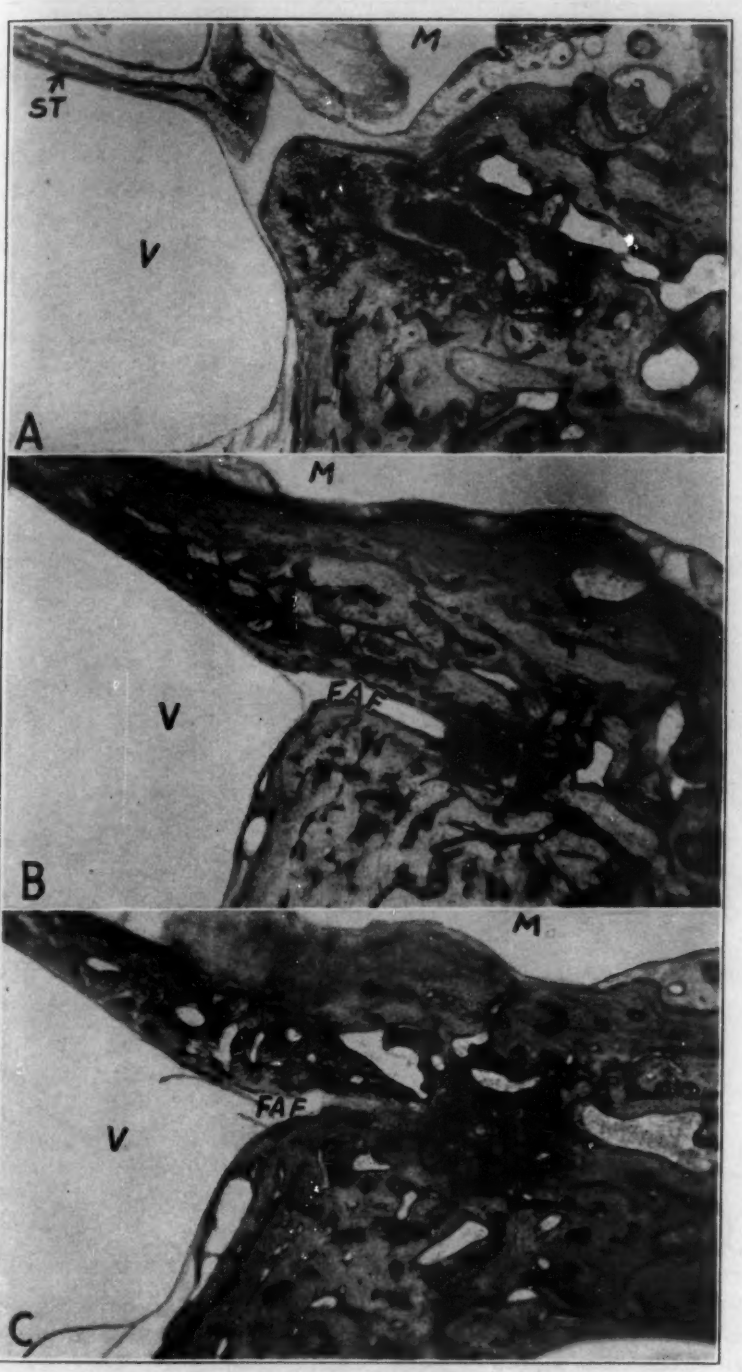

Fig. 3.—*A*, baby 82, aged 3 years (slide 30, section 6); *B*, baby 96 L, aged 3 years (slide 39, section 4); *C*, baby 83, aged 10 weeks (slide 43, section 2). In all three sections the fissula is partly or wholly obliterated by a gnarly mass of bone. In *A* there is still some cartilage at the center of this area.

M is middle ear; *St*, stapes; *V*, vestibule, and *F A F*, fissula ante fenestram.

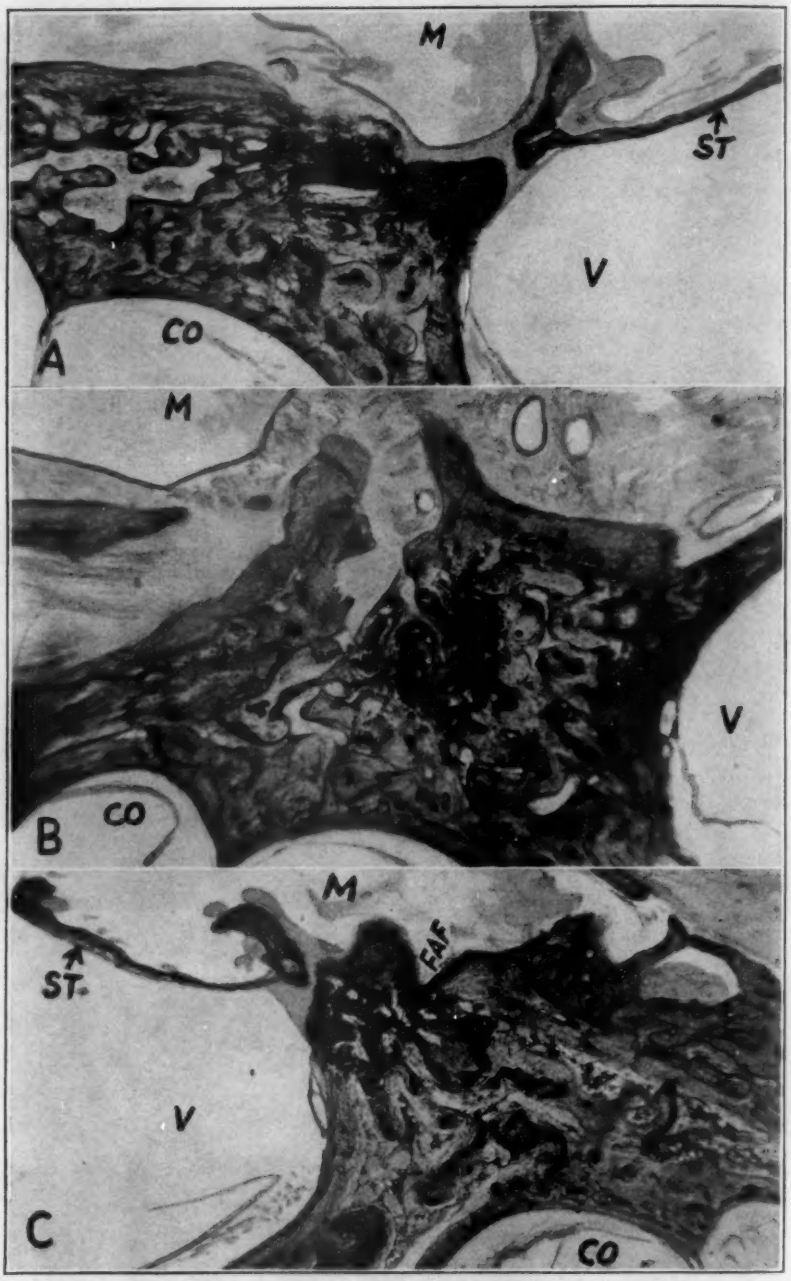

Fig. 4.—*A*, baby 93 R, aged 14 months (slide 30, section 4); *B*, baby 93 R, aged 14 months (slide 33, section 6); *C*, baby 79, aged 5 days (slide 28, section 4). In all three sections a patch of gnarly, sclerotic-appearing bone lies in the region of the fissula.

M indicates middle ear; *St*, stapes; *V*, vestibule; *Co*, cochlea, and *F A F*, fissula ante fenestram.

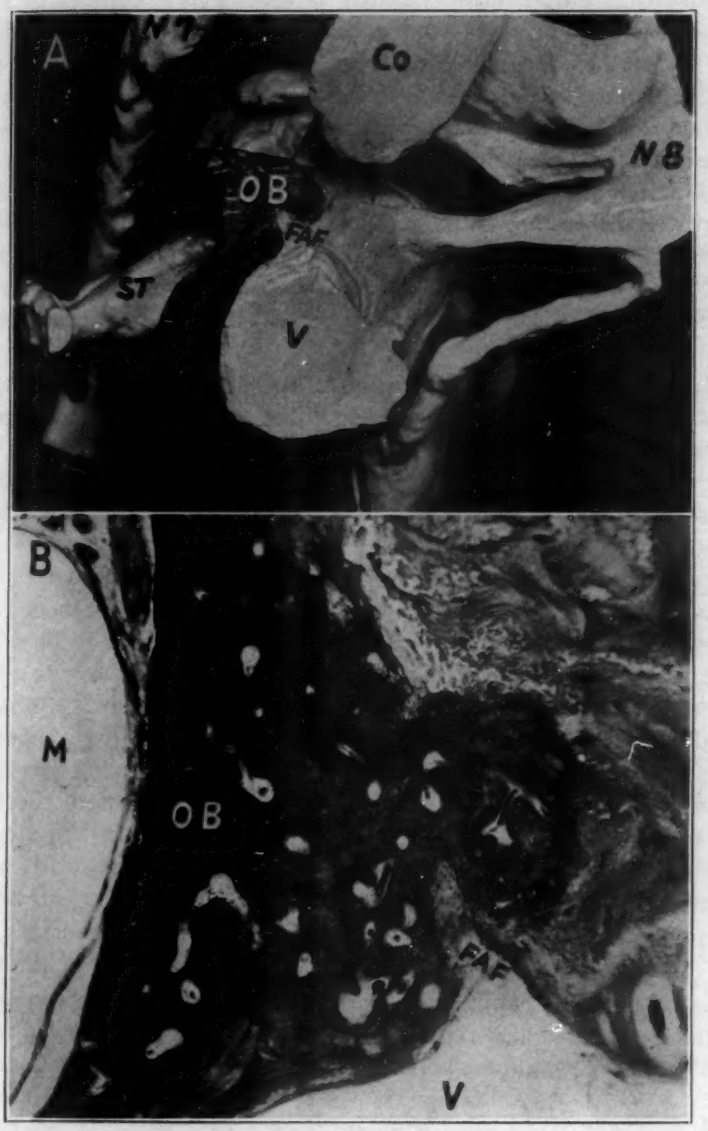

Fig. 5.—*A*, model of part of the internal ear, showing an otosclerotic area of bone (*O B*). The area lies anterior to the stapes (*S T*) and caps the vestibular end of the fissula ante fenestram (*F A F*).

B, section through the otosclerotic area shown in *A*. The dark bone (*O B*) is sclerotic. It surrounds the fissula ante fenestram (*F A F*). *M* indicates middle ear, and *V*, vestibule.

The serial sections from which the model (*A*) was made and the photograph (*B*) was taken belong to Dr. J. G. Wilson, who loaned them to me for study.

determined. I have made vascular injections in a number of human fetuses and have obtained some good injections of the vessels of the ear, but not of the spaces in the fissula. This suggests that these spaces may be lymphatic. Further observations are necessary. 3. The normal appearance of the fissula is shown in figure 1 *A* and *B*. This normal appearance occurred in about 66 per cent of the ears of all fetuses and children studied. In fetuses, in which the fissula is unusually large or in which there are outpocketings from it, secondary cartilage formations commonly occur, apparently for the purpose of limiting the fissula (fig. 2 *B*). This condition occurred in about 34 per cent of the ears studied. Late in fetal life or early in childhood these cartilages undergo ossification.

In some cases these cartilage masses become unusually large and tend to obliterate parts of the fissula (figs. 2 *A* and 3 *A*). Such large cartilage masses may not ossify until after birth, and some have been noted in children 5 years old. When ossification occurs in such cartilage masses, a gnarly, unstable bone results (fig. 3 *B* and *C*). Late attempts at bone formation in such areas result in a bone that resembles otosclerotic bone (fig. 4 *A, B* and *C*). While I do not have material from older children to show what happens to such unstable areas, there is some evidence that later attempts to rebuild them may result in otosclerotic bone and that such rebuilding may extend to involve the entire fissula and the stapes joint. 4. It is significant that, although sclerotic bone usually arises in the fissular region, the fissular tissue itself is resistant to the process. The otosclerotic patch may become rather large, even to the extent of involving much of the stapes joint before the fissula becomes obliterated. This is shown in figure 5 *A* and *B*. Here the otosclerotic patch is large, but the vestibular end of the fissula is not yet affected. This indicates that the fissula may not become obliterated by the process until the new bone has spread to involve the stapes joint. The question arises: Might it not also be the occlusion of the fissula that contributes to hardness of hearing, instead of stapes fixation alone? Certainly, some other explanation is in order, since hearing returns after fenestration, although the stapes is still fixed.

THE PERIOTIC LABYRINTH

As far as is known, the periotic labyrinth is a closed tissue space except that it communicates with the arachnoid spaces through the aquaeductus cochleae. This aqueduct opens into the beginning of the scala tympani near the round window (fig. 6). This is one end of the perilymphatic system. If the aquaeductus cochleae is the inlet of fluid from the cerebrospinal spaces, as is commonly supposed, then all of the periotic labyrinth is a blind, tortuous space. Is there no place for drain-

age of the perilymph from this labyrinth? If not, why does it not become stagnant? The fissula ante fenestram is a structure which could well serve this purpose. It is situated in the midportion of the labyrinth. Typical periotic tissue extends into it from the vestibule and communicates with the mucoperiosteal membrane of the middle ear. Definite endothelium-lined spaces are contained within the fissular tissue. In short, the structure and position of the fissula ante fenestram give one the impression that it may be a lymphatic filtration area, similar to that of the filtration angle of the eye. If this is the case, then obliteration of the fissula by otosclerotic bone would result in stagnation of the perilymphatic fluid. Fenestration could relieve this stagnation. Certainly,

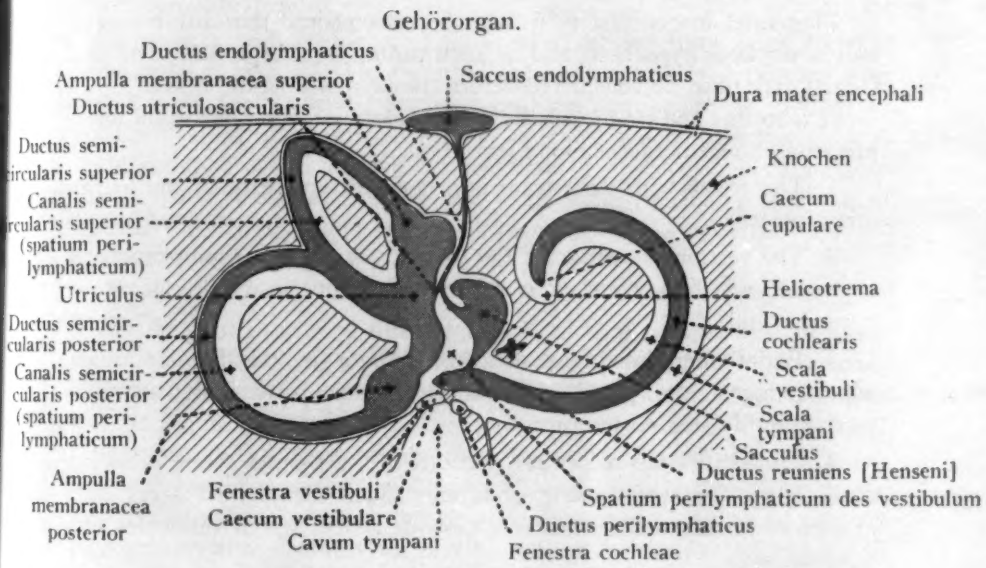

Fig. 6.—Spalteholz' (Handatlas der Anatomie des Menschen, ed. 7, Leipzig, S. Hirzel, 1914, p. 825) diagram of the otic labyrinth. Note that the ductus perilymphaticus (aquaeductus cochleae) opens into the blind end of the scala tympana. This is at one end of the perilymphatic (periotic) labyrinth. The + indicates the point where the fissula ante fenestram communicates with the vestibule.

Jenkins had some such idea when he made holes in the otic capsule in order to produce a change in the density of the perilymph.

I am well aware of objections to this interpretation, which I present only as a suggestion. Objections can be raised to any theory relating to the ear that is extant today. I am merely suggesting that one consider fairly all possibilites.

One of the main objections to considering the fissula as a filtration area is that in other animals there is no fissula and yet they hear. This

is a legitimate objection which has not been answered. It can only be stated that the fissula in the human periotic capsule is a constant and phylogenetically new structure and as such one would expect it to have a definite function.

Another objection comes from the adherents to the theory that two windows in the perilymphatic wall are necessary for sound conduction. According to them, fenestration creates a new window in place of the oval window, which was blocked when the stapes became fixed. But this two window theory is also open to question, for, according to Pohlman's interpretation, if two similar membranous windows conduct sound to the perilymph from opposite directions, then the sound waves should be neutralized.

Therefore, in conclusion, it must be remembered that any theory is only a working hypothesis and as such cannot be accepted as fact. All factual data must be considered before the final word can be said.

It is to this end that I submit for consideration these facts and interpretations about the fissula ante fenestram.

1. The fissula ante fenestram is a constant structure in the human otic capsule.

2. The vestibular portion of the fissula contains a perilymphatic type of tissue, which is continuous with the mucoperiosteal tissue at its tympanic end.

3. Endothelium-lined channels are present in the fissular tissue, which suggest the possibility that the fissula ante fenestram is a perilymph-filtering structure.

4. Chondrification of certain portions of the fissular tissue occurs in some fetuses or very young children. Such chondrified areas later change to an atypical or unstable osseous tissue, which resembles otosclerotic bone.

5. The fissular tissue itself is resistant to ossification, but with the increase of otosclerotic bone it gradually becomes obliterated. Such obliteration would destroy any function which the fissula would normally subserve.

6. If the fissula is a perilymph drainage area, then its obliteration would result in stagnation of the perilymph. Fenestration could reestablish perilymph drainage.

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ASSOCIATION OF NERVE DEAFNESS AND RETINITIS PIGMENTOSA

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DANVILLE, VA.

The association of nerve deafness and pigmentary degeneration of the retina is well known. A study of this combination of hereditary defects from an otologic point of view has not been found in the American literature.

The first audiometric studies on this subject have been recently published by Hardy.[1] He recorded the audiograms of 3 persons with retinitis pigmentosa and associated deafness, but made no attempt to consider the otitic aspects in greater detail. The audiograms showed a similar type of nerve deafness, decrease or absence of bone conduction, a positive Rinne reaction and a gradual progressive decrease in hearing for the successive higher frequencies.

GENETIC ASPECTS

In the first decade of this century Nettleship pointed out the association of retinitis pigmentosa with deafness, deaf-mutism and other defects of the central nervous system. Since that time many references can be found in the literature concerning the association of retinitis pigmentosa and deafness, chiefly from the point of view of the ophthalmologist or the eugenist. The largest and most authoritative study was made by Bell.[2] Among her pedigrees she found 96 cases of retinitis pigmentosa with deafness or mutism out of a total of 919 cases of retinitis pigmentosa, or an incidence of 10.4 per cent. In the pedigrees in which deafness was associated with retinitis pigmentosa 74.3 per cent were either deaf or deaf-mutes. Bell stated:

> . . . where deafness occurs in relationship with retinitis pigmentosa in any member of a stock this defect will almost invariably be associated with any case of retinitis pigmentosa that occurs in the same sibship and will tend to be associated also with cases which occur in other sibships of the stock.

Presented as a candidate's thesis to the American Laryngological, Rhinological and Otological Society, Inc., 1941.

1. Hardy, G.: Deafness and Retinitis Pigmentosa, Am. J. Ophth. **23**:315-317 (March) 1940.

2. Bell, J.: Retinitis Pigmentosa and Allied Diseases: Congenital Stationary Night-Blindness, Glioma Retinae, in Treasury of Human Inheritance, University of London, Francis Galton Laboratory for National Eugenics, Eugenical Laboratory Memoirs 21, 1922, vol. 2, pt. 1.

In the Usher pedigrees, only the pedigrees in which 1 case of deafness existed as an associated defect being considered, all the persons with retinitis pigmentosa from such pedigrees were deaf. Bell remarked that there are a few cases on record in which persons with this combination of defects have married and have been reported to have normal children. Nettleship described a case of retinitis pigmentosa in a deaf woman who was married to an unrelated, normal man. Two children resulted from this union, a normal son and a daughter with retinitis pigmentosa. Retinitis pigmentosa with deafness is inherited as a recessive, since in 40.2 per cent of cases the parents are consanguineous. In 33 per cent of Nettleship's cases of retinitis pigmentosa deafness was present, and in 4 per cent of cases of deaf-mutism out of 1,225 institutional cases retinitis pigmentosa existed.

Of the etiologic factors in congenital deafness and deaf-mutism, heredity and consanguinity provide the only basis for study; of 1,928 cases of congenital deafness, these two factors were present in 723.

In a study of retinitis pigmentosa and associated neuropsychiatric changes, Wortis and Shaskan [3] found retinitis pigmentosa associated with deafness in 44 per cent of their cases.

HISTOLOGIC CHANGES IN HEREDITARY DEAFNESS

Many reports have accumulated in the literature on the histologic changes observed in cases of hereditary deafness or deaf-mutism. The majority of these reports emphasize that the lesions are postinflammatory or secondary to increased labyrinthine pressure. From an anatomic and histologic point of view, it is apparently difficult to decide whether the change should be placed on a congenital or an acquired basis. Wittmaack added to this difficulty by reproducing experimentally similar histologic changes in the membranous cochlea which heretofore had been regarded as congenital, especially in congenital deaf-mutism, a disease undoubtedly hereditary. He expressed the opinion that, aside from deaf-mutism of inflammatory origin, in the majority of cases of congenital deaf-mutism the condition is caused by hydrops of the labyrinth during birth, followed by posthydropic degeneration of the organ of Corti and of the neurons belonging to it. Deafness caused by toxic irritants and disturbances of development is less frequent.

Unable to discard the hereditary feature in these cases, some authors have explained the histologic changes taking place as secondary to an inherited weakness of the inner ear, which is unable to withstand the ravages of adjacent inflammation or increased labyrinthine pressure at

3. Wortis, S. B., and Shaskan, D.: Retinitis Pigmentosa and Associated Neuropsychiatric Changes, J. A. M. A. **114**:1990-1991 (May 18) 1940.

birth. Uffenorde contended, however, that the proof of heredity must lie in the family history alone.

According to Altmann and Hager,[4] the histologic diagnosis of congenital deafness is on firmer ground in the presence of other malformations of the ear. The malformations observed in the bony framework of the cochlea, and associated with extensive changes of the cochlear duct, especially of the epithelial parts, are known as malformations of the Mondini type. Absence or defective structure, atresia of the cochlear duct, complete absence or defective structure of the organ of Corti in parts of the duct may occur. The organ of Corti may be reduced to a flat mound of undifferentiated cells, corresponding to an early stage in the embryonic development of the organ; this picture may appear, however, in secondary changes of posthydropic neuroepithelial degeneration or simple secondary atrophy.

Altmann and Hager pointed out that malformations of the Mondini type are present in cases of the so-called hereditary degenerative type of hardness of hearing and of deafness of the inner ear type, which is said to be inherited as a dominant hereditary trait. In cases of so-called sporadic deaf-mutism, which is inherited in a recessive manner, there is defective development of the nervous sound-perceiving apparatus, with secondary degenerative changes of the nerve substance of the auditory nerve, including Corti's organ. In some cases there are also changes in the bony structure, in the septums of the scala and in the lamina spiralis ossea.

Altmann and Hager [4] stated that changes which were considered as probably congenital were observed in a case of deaf-mutism associated with retinitis pigmentosa, as described by Siebenmann and Bing.

> Corti's organ is absent in the lower basal coil and begins in the upper basal coil as a hill consisting of cuboidal cells with fine granular protoplasm. This hill is situated not in its typical place but is located laterally so that it lies almost completely outside of the tectorial membrane. Beginning in the middle turn, the ridge gradually assumes a more normal position as it ascends into the uppermost turn. The spiral ganglion is strikingly poor in cells. In the maculae and cristae only slight degenerative changes could be found.

Similar changes are found in the waltzing mice, a condition which is also inherited as a simple mendelian recessive characteristic. The deafness is said to be based on a congenitally defective anlage associated with secondary degenerative processes; there is atrophy of the branches, ganglions and roots of the eighth nerve, especially the cochlear nerve, with degenerative atrophic changes of the epithelium of Corti's organ and the macula sacculi, together with defective formation of the stria

4. Altmann, F., and Hager, C. E., Jr.: Congenital Malformations of the Ear, in Fowler, E. P., Jr.: Medicine of the Ear, New York, Thomas Nelson & Sons, 1939.

vascularis. In the waltzing mouse, and also in the waltzing guinea pig, the circling is of central nervous system origin; the vestibular end organs are normal.

In his study on the waltzing guinea pig, Lurie [5] pointed out that the interesting changes observed in the membranous cochlea of such an animal illustrates how nature causes deafness of the inherited type and also the way in which the organ of Corti may degenerate as a result of disease or toxic substances. He concluded that the deafness of the waltzing guinea pig is caused by degeneration of the organ of Corti. The degeneration occurs in a definite way, the external hair cells being involved first and the internal hair cells later; the cochlear nerve and the spiral ganglion cells degenerate at a later period.

Altmann and Hager remarked that sporadic deaf-mutism associated with retinitis pigmentosa is of rather frequent occurrence. They stated that one usually finds in cases of this condition diminution or absence of irritability of the vestibular apparatus.

RETINITIS PIGMENTOSA

Duke-Elder [6] stated that retinitis pigmentosa is a disease of unknown cause, with a decided hereditary tendency. Pathologically, it is a degeneration of the retinal neuroepithelium, primarily the rods, and ophthalmoscopically the main features are attenuation of the retinal vessels, optic nerve atrophy and widespread pigmentary changes in the retina. Pigmentary degeneration of the retina is inherited in a dominant, recessive or sex-linked manner, although Macklin [7] stated that eleven types of inheritance exist.

Wibant differentiated two types of retinitis pigmentosa. The dominant type is unassociated with any other lesion, and the recessive type is usually accompanied by deafness or other nerve lesion. Apparently, one must deal with many variables when studying the genetics of retinitis pigmentosa.

Besides deafness, myopia, mutism, mental deficiency, epilepsy, hereditary ataxia and hereditary spastic paraplegia are associated with retinitis pigmentosa. The association of retinitis pigmentosa with pituitary and skeletal abnormalities, obesity, polydactyly, hypogenitalism and mental retardation is known as the Laurence-Moon-Biedl syndrome. The condition is familial, the inheritance being of the recessive mendelian type.

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5. Lurie, M. H.: The Waltzing (Circling) Guinea Pig, Ann. Otol., Rhin. & Laryng. **50**:113-127 (March) 1941.

6. Duke-Elder, W. S.: Textbook of Ophthalmology, St. Louis, C. V. Mosby Company, 1941, vol. 3.

7. Macklin, M. T.: The Role of Heredity in Disease, Medicine **14**:1-75, 1935.

The disease is slow, chronic and progressive, usually ending in blindness. The first symptoms, night blindness and limitation of the visual field, are noted in the early teens.

According to Duke-Elder, the most widely accepted theory relative to the etiology of retinitis pigmentosa is that credited to Collins; he expressed the belief that the disease is a primary and inherent process of the nature of an abiotrophy, a premature senility and death of the cells of a specified tissue. He assumed that the associated phenomena of deafness and nervous disturbances were due to a similar abiotrophic process in the ears and the central nervous system. It has been proposed that two types of retinitis pigmentosa exist, one retinal and ectodermal and the other choroidal and mesodermal; the pathologic changes in the inner ear would be associated more closely with the retinal form.

REPORT OF CASES

The family studied consisted of 11 members: the father, the mother, 8 sons and 1 daughter. Four of the sons had undoubted retinitis pigmentosa and difficulty in hearing. The mother and daughter were well and showed no evidence of disease of the ears or eyes. The father was feeble; no studies could be carried out on him. He saw well, however, but had an impediment in speech (otogenic?) similar to that of the 4 sons with nerve deafness. There were 35 grandchildren, varying in age from several months to 20 years. No evidence or symptoms of disease of the eyes or ears was elicited from the history or from the persons examined. The mother's father and mother were first cousins. A first cousin on the father's side was a deaf-mute, but the condition was found to be acquired. Another first cousin on the paternal side was said to have difficulty in seeing and hearing; this person could not be contacted. The family was from a lower stratum of society.

In figure 1 are recorded the audiograms of the 4 affected brothers, ranging from the youngest to the eldest. A similar type of nerve deafness is noted, with gradual progressive decrease in hearing for the successive higher frequencies. The bone conduction findings are indicated according to Fowler. Masking was not employed because of the similar loss of hearing in each ear. A quiet, but not a soundproof, room was used.

Figure 2 is the interesting audiogram of a brother who showed no evidence of retinitis pigmentosa.

The 4 afflicted brothers varied in age from 25 to 43 years. They had always had difficulty in hearing, and since childhood had found it impossible to see after dark. All had greatly impaired vision, ranging from 5/200 to 20/30. All visual fields were constricted; no ring scotoma could be demonstrated. Figure 3 illustrates the visual fields of 1 of the brothers. There was no history of acute aural disease or severe childhood infection. The patients complained occasionally of tinnitus.

A more detailed report on the eldest brother will suffice; the others are similar except for less pronounced visual impairment. His vision was reduced to 2/200 and 5/200 and was not improved by glasses. The Wassermann reaction was negative, and general physical examination revealed nothing abnormal. As with the other brothers, he had a pronounced speech defect. Tuning fork tests showed a notable diminution in bone conduction for the c and c-1 forks, with absence of

bone conduction for the c-2 and c-3 forks; the c-4 fork was not heard at all. The Rinne reaction was positive. The eyes showed posterior polar cataracts, pronounced pallor of the optic disk, greatly attenuated retinal arterioles and areas of retinal pigmentation scattered over the entire fundus. It was impossible to obtain visual fields.

Examination of the nose, sinuses, epipharynx, ears, mouth and throat showed no abnormality. The tonsils had been removed, and the tympanic membranes appeared normal; the teeth were in good repair, and both eustachian tubes were patent. Vestibular stimulation by the caloric method (cold water at 68 F.) showed a normal reaction in each ear.

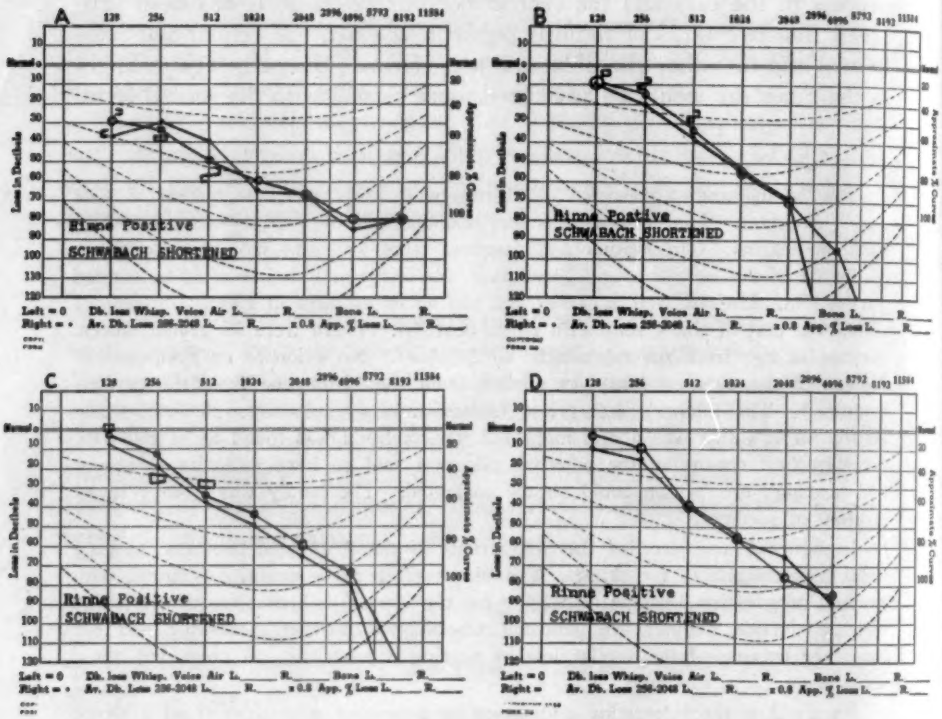

Fig. 1.—Audiograms illustrating nerve deafness in 4 brothers with retinitis pigmentosa.

A, T. M., aged 43; vision 2/200 and 5/200.

B, A. M., aged 38; vision 20/200 and 20/200, with corrected vision 20/40 — 1 and 20/30 — 2.

C, J. M., aged 26; vision 20/40 + 3 and 20/25 — 4.

D, R. M., aged 36; vision 20/200 + 2 and 20/200.

One of the afflicted brothers was married but had separated from his wife; his 2 children, a boy and a girl, aged 7 and 9 years, were not available for examination. They were said to hear and see well. Burks [8] expressed the opinion

8. Burks, B. S.: Personal communications to the author.

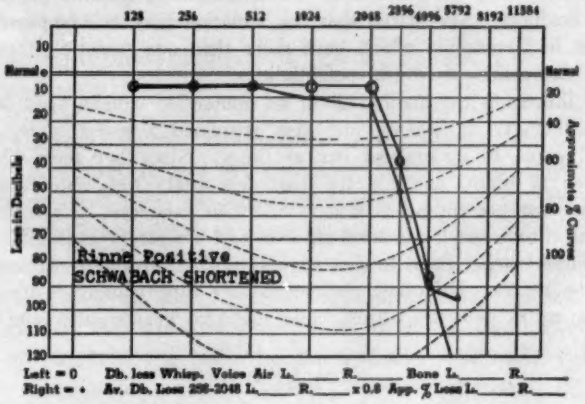

Fig. 2.—Audiogram of fifth brother, aged 32, who does not have retinitis pigmentosa. Vision 20/20 and 20/20.

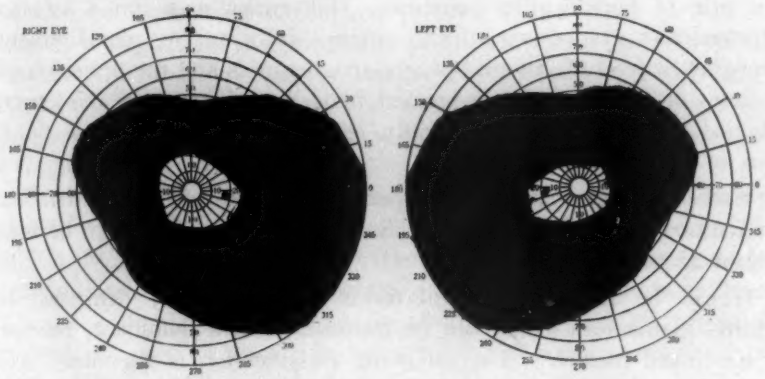

Fig. 3.—Visual fields of R. M., aged 36.

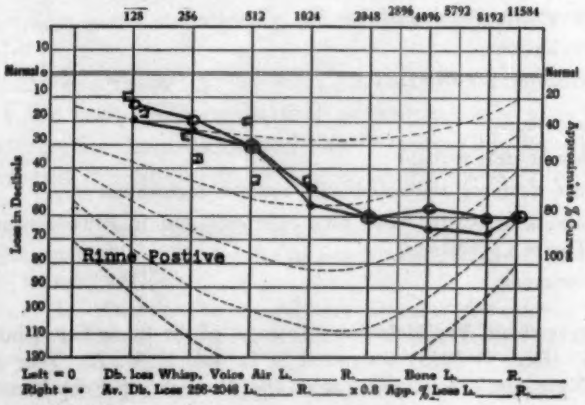

Fig. 4.—Audiogram of L. E., aged 30, with difficulty in hearing and retinitis pigmentosa.

that the condition here described is a recessive form of retinitis pigmentosa associated with deafness. She stated that the situation may be compared to that of certain genes in Drosophila which have more than one somatic effect and variability in the expression of several potentialities.

Figure 4 illustrates the audiogram of an unmarried woman aged 30. She had always had difficulty in hearing and also possessed a speech defect, making it difficult for people to understand her at times. Since her teens she had been unable to see at night, and for the past five years her vision has gradually decreased, until at present her vision is 5/200 in each eye. Examination of the ears, nose and throat and the general physical and laboratory examinations revealed nothing of importance. Each fundus showed typical advanced pigmentary degeneration of the retina. No other member of her large family is known to have impaired hearing or defective vision. Her maternal grandmother and father were first cousins.

COMMENT

Hammerschlag [9] placed the various hereditary diseases of the inner ear—hereditary degenerative total deafness, progressive labyrinthine deafness, premature presbyacusia and otosclerosis—under the all-inclusive title of heredopathia acustica. This entity was stated to show pathologically graded variations, ranging from serious developmental anomalies of fetal life to mild progressive deafness and the "physiologic" deafness of old age. He expressed himself in favor of a monomeric, polyallelic heredity, which logically explains the heredopathia on the basis of a single mutated gene.

Escheweiler estimated that 50 per cent of cases of deaf-mutism have a hereditary basis, although unquestioned proof of heredity can be found only in 25 to 30 per cent.

It can be stated in general terms that hereditary deafness, like retinitis pigmentosa alone, can be transmitted in a dominant, recessive or sex-linked manner. The following classification is feasible.

Dominant heredity

1. Progressive labyrinthine deafness
2. Degenerative total deafness
3. Otosclerosis
4. Premature presbyacusia

Recessive heredity

1. Sporadic deaf-mutism
2. Nerve deafness associated with retinitis pigmentosa
3. Deafness associated with blue sclerotics and fragilitas ossium (van der Hoeve's syndrome)
4. Otosclerosis

9. Hammerschlag, V.: Hereditary Diseases of the Inner Ear, Klin. Wchnschr. **12**:1903-1906 (Dec. 9) 1933; abstracted in Brown, E. V. L.: Year Book of the Eye, Ear, Nose and Throat, Chicago, The Year Book Publishers, Inc., 1934, p. 406.

Sex-linked heredity

1. Progressive nerve deafness, as reported by Ersner [10]
2. Otosclerosis

The appearance of of nerve deafness in combination with a disease commonly considered hereditary provides material for discussion whether the anomalies have their origin from a single defect in the germ plasm or arise as hereditary diseases existing side by side. Weber [11] reviewed the factors cited in favor of a common origin of two hereditary disorders and those favoring an independent hereditary transmission. The combined appearance creates the impression that the two belong together, the more so since in the cases he studied the vestibular nerve did not respond to stimulation, a condition not present in the cases here reported. In favor of a separate transmission, it can be said that the two diseases are unalike, they usually appear separately and pigmentary degeneration of the retina is known to occur not only with recessive deaf-mutism but also with dominant hardness of hearing of the inner ear. Apparently, it is impossible to answer this question with the material at hand. Of interest is the audiogram of a brother who does not show any evidence of retinitis pigmentosa. There is a decided dip in hearing for the upper frequencies, although not as pronounced as in the audiograms of the afflicted brothers.

Burks [12] stated that possibly more than one gene for retinitis pigmentosa with deafness will eventually be demonstrated. The pedigrees with deafness show interesting variations; in some instances deafness is partial and in others it is complete, dating from birth or early infancy. In some pedigrees all the persons affected with retinitis pigmentosa are deaf and none of the unaffected are deaf, whereas in others deafness occasionally occurs in the unaffected as well as in the affected offspring, or even passes over some of the affected offspring.

Burks pointed out that in some families one may be dealing with two independent genes segregating for retinitis pigmentosa and deafness respectively.

Macklin [13] expressed the opinion that perhaps the same gene is responsible for both diseases, or that possibly closely associated genes have suffered some disturbance and one is responsible for the condition of the ears and the other for the defect in the eyes. She stated that,

10. Ersner, M. S., and Saltzman, M.: Progressive Analogous Nerve Deafness in Three Successive Generations with a Sex-Limited Inheritance, Laryngoscope **51**:241-245 (March) 1941.

11. Weber, H.: Combination of Pigment Degeneration of the Retina with Deafness, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **147**:1-68, 1940; abstracted, J. A. M. A. **114**:2155 (May 25) 1940.

12. Burks, B. S.: Retinitis Pigmentosa Tested for Incomplete Sex-Linkage, Eugen. News **22**:33-42 (June) 1937.

13. Macklin, M. T.: Personal communication to the author.

judging from the number of cases reported, this association did not seem unusual. Indeed, conversations with several otologists brought out the fact that they, too, have observed this association. Several years ago I endeavored to study a family of 8 children, 4 boys and 4 girls. The father and mother were first cousins. Three of the girls were deaf-mutes with retinitis pigmentosa, and the fourth was a feebleminded girl. The 4 brothers were said to be normal. One of the girls with retinitis pigmentosa and deaf-mutism married another deaf-mute; 2 children from this union were said to be normal. Difficulty in communication and reticence on the part of the patients illustrate the difficulty in attaining completeness in this type of survey.

In conclusion, it can be said that the cases under survey do not furnish the explanation of this combination of deafness. Genetics in the field of otology is complicated. More pedigrees are necessary to determine the exact role of heredity in otologic disease.

105 South Union Street.

REACTION OF MUCOUS MEMBRANES TO FIVE PER CENT SOLUTION OF SODIUM SULFATHIAZOLE

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Since the advent of the sulfonamide compounds there have been continual trial and error attempts to apply them to almost every kind of ailment. Local application has received much attention, and of late a much heralded article[1] on the use of sulfathiazole in the nose as a treatment and cure of chronic sinusitis has made me wonder just what effect it has on the nasal mucous membrane.

It has been proposed that a spray or instillation in the nose of a 5 per cent solution of sodium sulfathiazole (sodium salt of 2-[para-aminobenzenesulfonamido]-thiazole) three times a day for a number of days in one suffering from chronic sinusitis would cure the condition. This was based without presentation of experimental data; so there was no way to determine the effect of the drug on the mucous membrane. Then an article[2] came out telling of the very corrosive effect on the nasal mucous membrane of sulfathiazole instilled into the antrums and the nose. This was not corroborated by other cases; so it was difficult to substantiate this observation. It was mentioned in this article that the p_H of 5 per cent sodium sulfathiazole is about the same as that of arsphenamine, and since the violent reaction occurring in the tissues when arsphenamine escapes outside of a vein is known, there must be a similar effect on the mucous membranes of the nose and sinuses. This comparison did not seem to be exact because the sulfathiazole was not being injected under the mucous membrane.

This report is on the effect of sulfathiazole in different percentages instilled in noses of mice. Inbred Swiss mice were used, as developed by the Rockefeller foundation. The technic was the same as that pre-

Presented as a candidate's thesis to the American Laryngological, Rhinological and Otological Society, Inc., Oct. 30, 1941.

1. Turnbull, F. M.: Intranasal Therapy with Sodium Salt of Sulfathiazole in Chronic Sinusitis, J. A. M. A. **116**:1899 (April 26) 1941.

2. Fletcher, R.: Sodium Sulfathiazole: Its Caustic Action, California & West. Med. **55**:94 (Aug.) 1941.

viously used [3] in studying the effect of drugs on the upper and lower parts of the respiratory tract. The mouse is held firmly by putting the head into the large end of an ear speculum which has been cut in half. A drop of the medication is placed on the tip of the nose so as to cover the nostrils, and after a few seconds inspiration occurs and the medication is sucked into the nose. There is a good covering of the nasal cavity if the head is tipped back (if methylene blue is used and the head is split down through the septum immediately afterward the entire

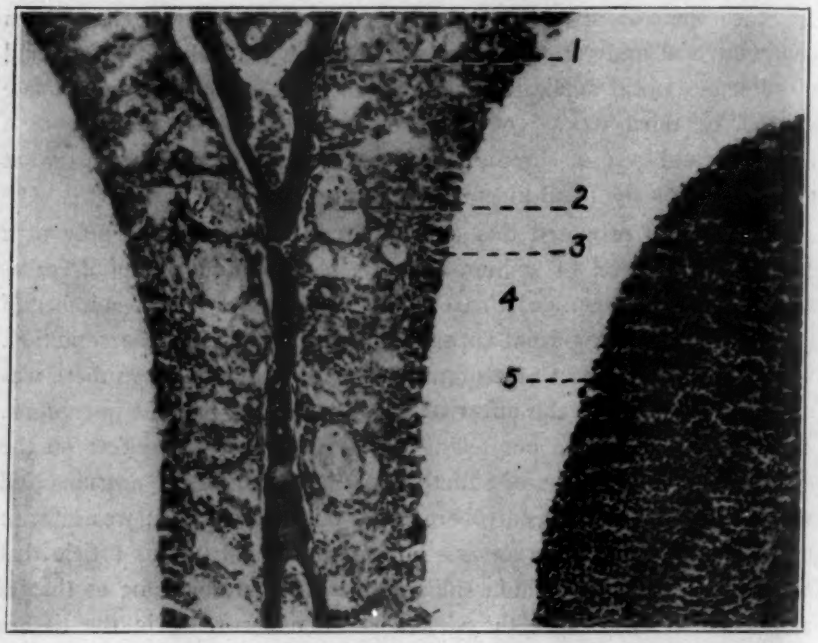

Fig. 1.—Transverse section through the nose, showing septum with olfactory mucous membrane. After one day of treatment (5 per cent sodium sulfathiazole in the mouse's nose three times a day) there is a large amount of purulent exudate and the epithelium of the olfactory mucous membrane is disorganized. Olfactory bundles are seen in the subepithelial layer. *1* indicates bone of the nasal septum; *2*, olfactory nerve bundle; *3*, olfactory epithelium, disorganized; *4*, nasal cavity; *5*, purulent exudate.

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3. Hunnicutt, L. G.: The Effect of Drugs on the Respiration of the Mucous Membrane System, read before the Eye, Ear, Nose and Throat Section at the Seventieth Annual Session of the California Medical Association, Del Monte, May 6, 1941; The Effect of Drugs on the Nasal Mucous Membrane, Tr. Pacific Coast Oto-Ophth. Soc. **26**:53, 1941.

lining of the nose, even down into the trachea, is seen to be stained with methylene blue).

The mice were treated with 0.5, 1 and 5 per cent solutions of sodium sulfathiazole. Since the 5 per cent solution has been recommended in literature, more extensive experiments were carried on with it. Instillations were made three times a day for fourteen days. In order to determine if there were any intermediary changes, 3 mice were killed every twenty-four hours, including the tenth day, and then a final group was killed on the fourteenth day. The lungs were saved and checked for

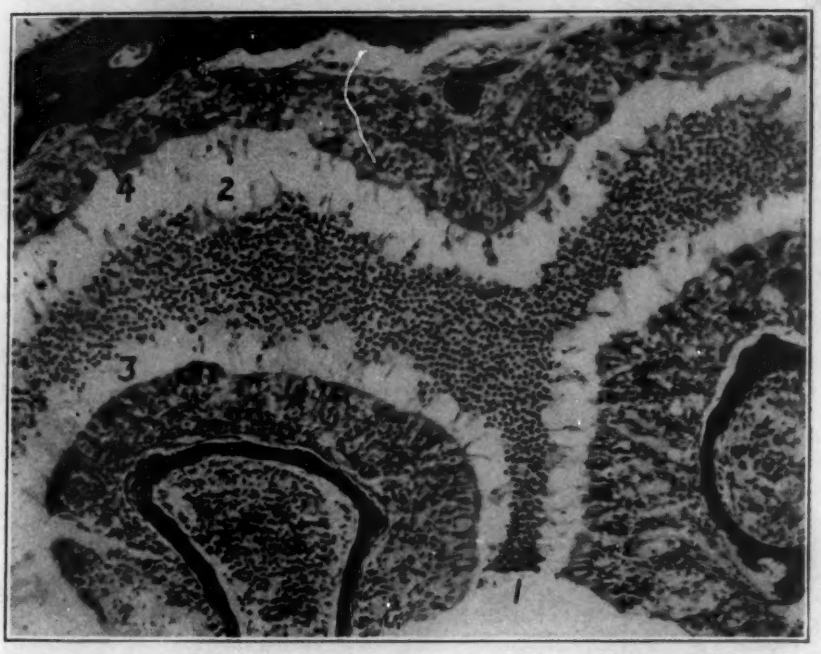

Fig. 2.—Section through the frontal sinus after two days of treatment (5 per cent sodium sulfathiazole in the mouse's nose three times a day), showing purulent exudate in the sinus and a marked inflammatory reaction in the mucous membrane. *1* indicates the frontal duct, opening from the nasal cavity; *2*, the frontal sinus cavity filled with purulent exudate; *3*, epithelial lining of the frontal sinus; *4*, frontal bone.

bronchopneumonia. Controls were obtained by using physiologic solution of sodium chloride in the same manner, and no changes were noted.

Purulent exudate was found, particularly during the first three days, in about half of the mice. After that the only finding was a slight excess of mucus. Apparently the mucous membrane becomes resistant to the irritating effect of the sulfathiazole. The most pronounced change was

in the olfactory epithelium, which was disorganized but not destroyed. The sinuses showed purulent exudate, and the epithelium, especially in the frontal sinuses, was highly inflamed. In this early group, in which a purulent exudate and inflamed mucous membrane were present, a number of the mice showed bronchopneumonia. Incidentally, these mice appeared sick, as noted by inactivity, poor eating and failure to keep their hair smooth.

It is well to note here that these changes in the respiratory mucous membrane system were identical with those seen after the use of shrink-

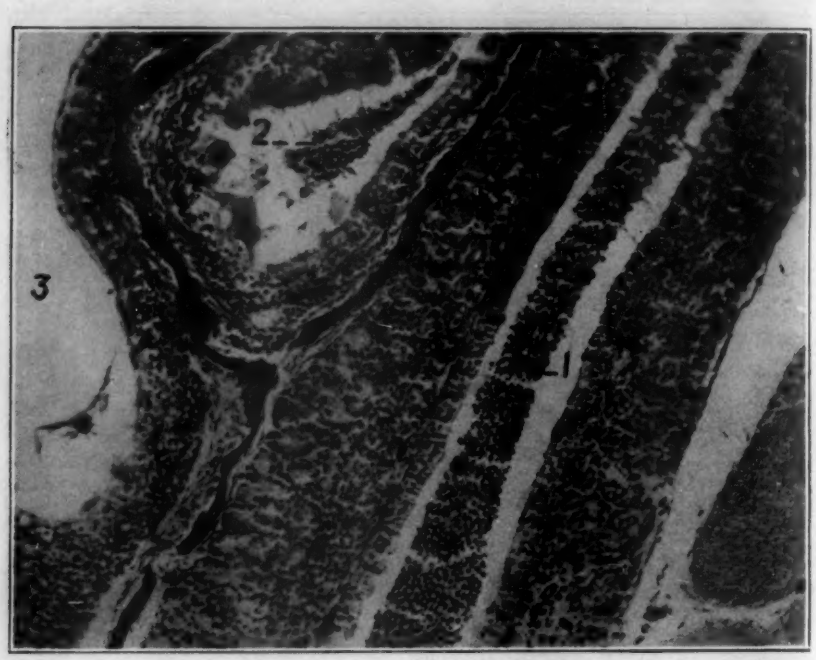

Fig. 3.—Section through the maxillary sinus after three days of treatment (5 per cent sodium sulfathiazole in the mouse's nose three times a day), showing purulent exudate in the sinus and behind the inferior turbinate (which turns up in the mouse). *1* indicates purulent exudate in the maxillary sinus; *2*, purulent exudate behind the turbinate; *3*, nasal space.

age medicines previously reported on.[3] In the report referred to, hypertonic solutions and many sclerotic drugs produced more or less the same local effect.

In the illustrations it will be noted that there was a marked inflammatory reaction for the first three days, and then the membrane returned

to practically normal and only a little excess mucus was seen on the epithelial surface. Even the controls had a little excess mucus; so one might say it was within normal limits.

Five-tenths and 1 per cent solutions of sodium sulfathiazole gave the same type of reactions when applied to the nasal mucous membranes, but the inflammatory reaction was in a lesser degree. Only illustrations of the use of the 5 per cent solution are presented, because that is the strength recommended by a writer and a drug company.

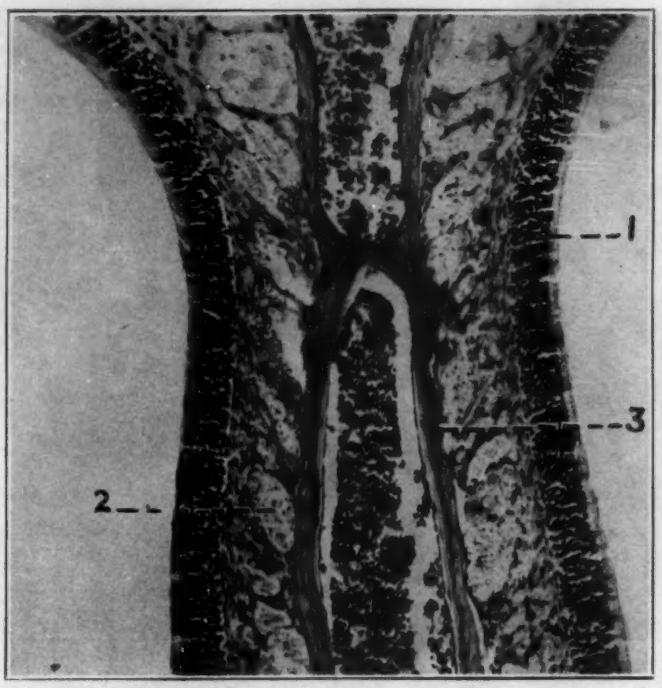

Fig. 4.—Section through the nasal septum after ten days of treatment (5 per cent sodium sulfathiazole in the mouse's nose three times a day), showing no change in the olfactory epithelium and no purulent exudate or excess mucus. *1* indicates olfactory epithelium which is normal; *2*, olfactory nerve bundles; *3*, bones of the nasal septum.

SUMMARY

Sodium sulfathiazole in a 5 per cent solution is an irritant to the nasal mucous membrane in the mouse and produces a purulent exudate and inflammation in the first three or four days of its use. Then, up through two weeks of its use, the inflammatory reaction subsides and almost no irritating effect is seen, as noted by lack of epithelial change

or purulent exudate. The olfactory mucous membrane, which is first affected when irritating drugs are used, as I had previously found, is not permanently injured. There is considerable spotty bronchopneumonia, just as I had found when the stronger nasal shrinkage medicines were used. This is apparently in direct relation to the amount of purulent exudate created in the nose.

The weaker solutions of sodium sulfathiazole also produce an inflammatory reaction and purulent exudate, but to a lesser degree.

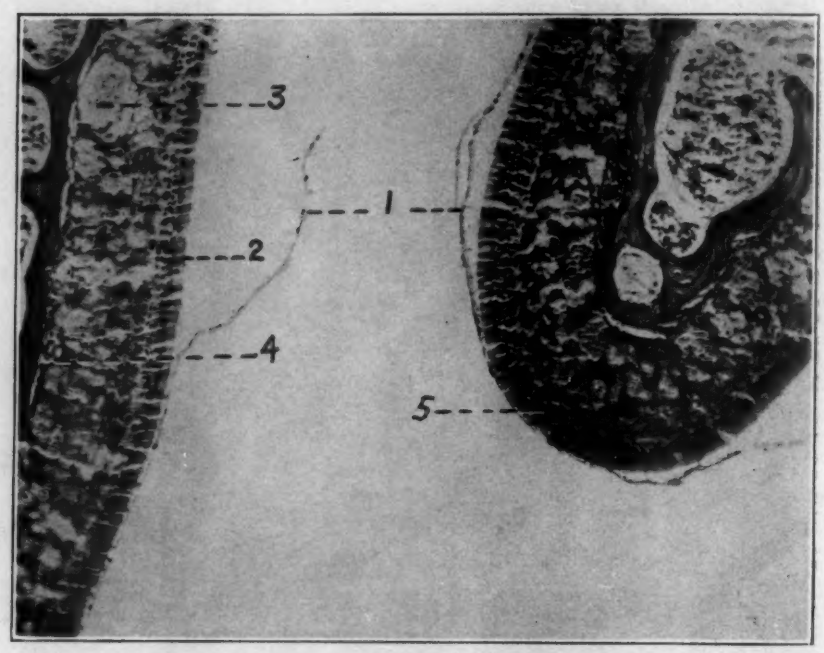

Fig. 5.—Section through the nasal septum, where the olfactory epithelium is merging into respiratory epithelium, and a turbinate, after fourteen days' treatment (5 per cent sodium sulfathiazole in the mouse's nose three times a day), showing only a slight excess of mucus. *1* indicates a small amount of mucus; *2*, olfactory epithelium; *3*, olfactory nerve bundles; *4*, septum; *5*, superior turbinate.

CONCLUSIONS

In the mouse there are no permanent ill effects from the use of a 5 per cent solution of sodium sulfathiazole. There is a marked inflammatory reaction the first few days, and after three days the effect on the mucosa is almost nil.

98 North Madison Avenue.

MICROSCOPIC EXAMINATION OF HUMAN LABYRINTHS FROM PATIENTS EXPOSED TO LOUD NOISES

DOROTHY WOLFF, PH.D.
ST. LOUIS

In the literature reports of traumatic or occupational deafness with descriptions of microscopic sections of the temporal bones are rare. Habermann [1] recorded the findings in the temporal bones of a 75 year old beggar who had been an iron-worker for twenty years. The man was killed by being run over by a train. Examination of the petrosas revealed that the stapes on one side was slightly displaced so that the anterior half was pulled out as from a great tug of the stapedius muscle. But the extreme age and violent death of this man obscure the proper interpretation.

At a later date Habermann [2] recorded the microscopic observations in 5 cases of what he termed "professional deafness." Two of these cases he illustrated with photomicrographs of the inner ear. He observed atrophy of the eighth nerve and of the organ of Corti. These cases also presented complications, tuberculous peritonitis in one and tabes dorsalis in the other.

Zange [3] illustrated his report of the case of a 29 year old shipbuilder. But this patient also had chronic tuberculosis, and it is known that defective hearing often develops in such patients.

Brühl [4] questioned the advisability of attempting to interpret degeneration of nerve fibers and ganglion cells seen in microscopic sections as being of traumatic origin. It is, of course, only from the observation of accumulated material with similar histories that accurate conclusions can be drawn. Routine autopsies afford the only means of acquiring data of this type on the human ear.

The petrosas from 3 patients exposed to loud noises were obtained in routine autopsy collections at the Washington University Medical School.

From the Oscar Johnson Institute, Washington University Medical School.

1. Habermann, J.: Ueber die Schwerhörigkeit der Kesselschmiede, Arch. f. Ohrenh. **30**:1-25, 1890.

2. Habermann, J.: Beiträge zur Lehre von der professionellen Schwerhörigkeit, Arch. f. Orhenh. **69**:106-130, 1906.

3. Zange, J.: Beitrag zur Pathologie der professionellen Schwerhörigkeit, Arch. f. Ohrenh. **86**:167-174, 1911.

4. Brühl, G.: Beiträge zur pathologischen Anatomie des Gehörorgans, Ztschr. f. Ohrenh. **52**:232-292, 1906.

Fig. 1 (case 1).—The left cochlea of a woman who shot herself in the right temple, the wound of exit being in the left temporal lobe. Note the intact vestibular (Reissner's) membrane.

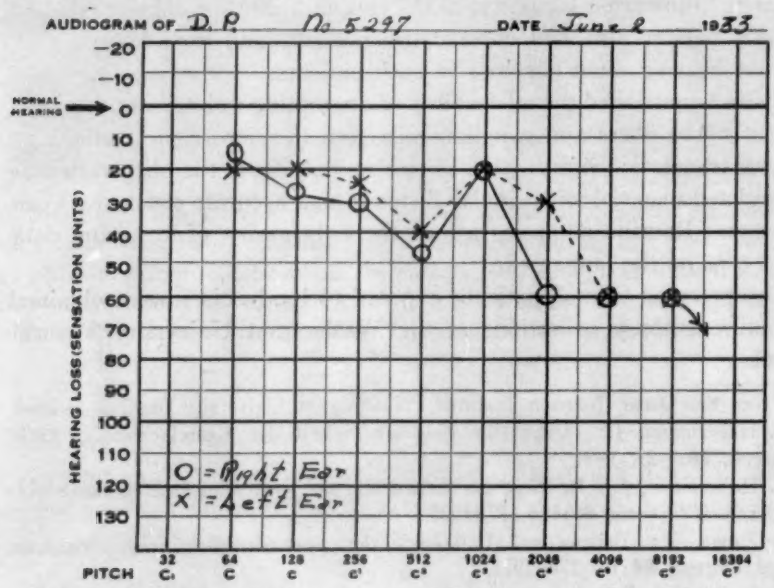

Fig. 2 (case 2).—Audiometer record of the patient with the bilateral fracture of the stapes illustrated in figure 3.

REPORT OF CASES

CASE 1.—A 29 year old white woman shot herself in the right temporal region. The wound of exit was in the left temporal region. The left petrosa

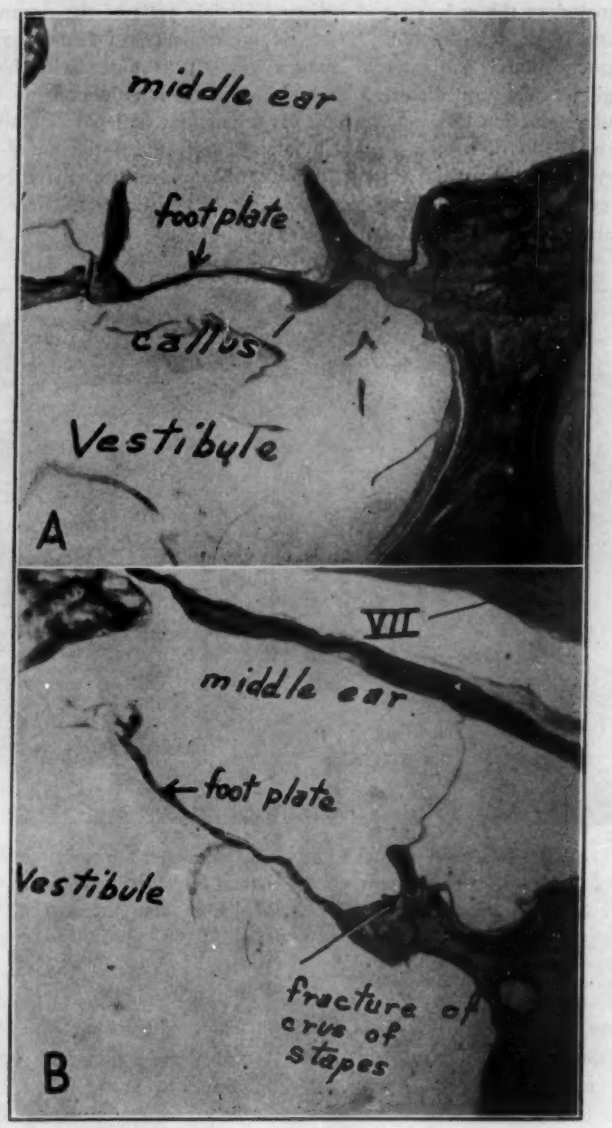

Fig. 3 (case 2).—*A*, callus from a fracture of the anterior portion of the foot plate of the stapes in the left ear of the riveter. *B*, fracture of the posterior crus of the right stapes of this patient.

only was intact and could be procured at autopsy. Figure 1 illustrates the cochlea of this patient. It is interesting to note that the delicate vestibular (Reissner's) membrane is intact from base to apex. Careful examination of the

microscopic sections reveals one small tear in the upper apical coil. This, however, may be an artefact since the block had to be reinfiltrated.

The organ of Corti is in situ but not normal. One cannot say how much damage was done at the time of the blast. The tectorium appears as a thin line agglutinated to the reduced and indistinguishable cells of the organ of Corti. Tunnel space is not identifiable. There is no evidence of hemorrhage within the inner ear, although the internal auditory meatus is laden with blood. The spiral ganglion cells are distorted and show marked reduction of cytoplasm. The vestibular ganglion cells appear unusually granular and pale staining. Some of these are devoid of cytoplasm. The longitudinal cut of the nerve bundle in the auditory meatus presents a somewhat mottled appearance, there being areas where no dye has been taken up in the course of the nerve. These are interspersed with lengths of normal-staining nerve.

Fig. 4 (case 2).—*A*, section of the inner ear. Reissner's membrane is not in situ in any turn. The organ of Corti is not normal in any turn. Note the unexplained location of the basilar membrane in the apical coil, also the marked atrophy of the spiral ganglion cells in the middle coil. *B* and *C*, on oppositie page, show details at high magnification.

The foot plate of the stapes is in situ. The malleoincudal joint is in situ. The attachment of the incus to the stapes is not found. Only the upper half of the drum membrane is in situ. It is impossible to know how much of the loss of the lower half is due to the blast and how much to difficulty in removing the block. It is evident that the pathologic condition of the membrane in the intact portion is the result of the blast, however. In the area over the attachment to the malleus the epidermis is widely separated from the fibrous and mucosal layers. The intervening space is packed with free blood cells. Near the superior border of attachment the epithelial lining is widely separated from the fibrous layer, and here the intervening space is packed with free blood cells.

These findings are probably related to the arrangement of blood supply in the region.

Free blood is present in some of the parantral cells and in the niches of the windows.

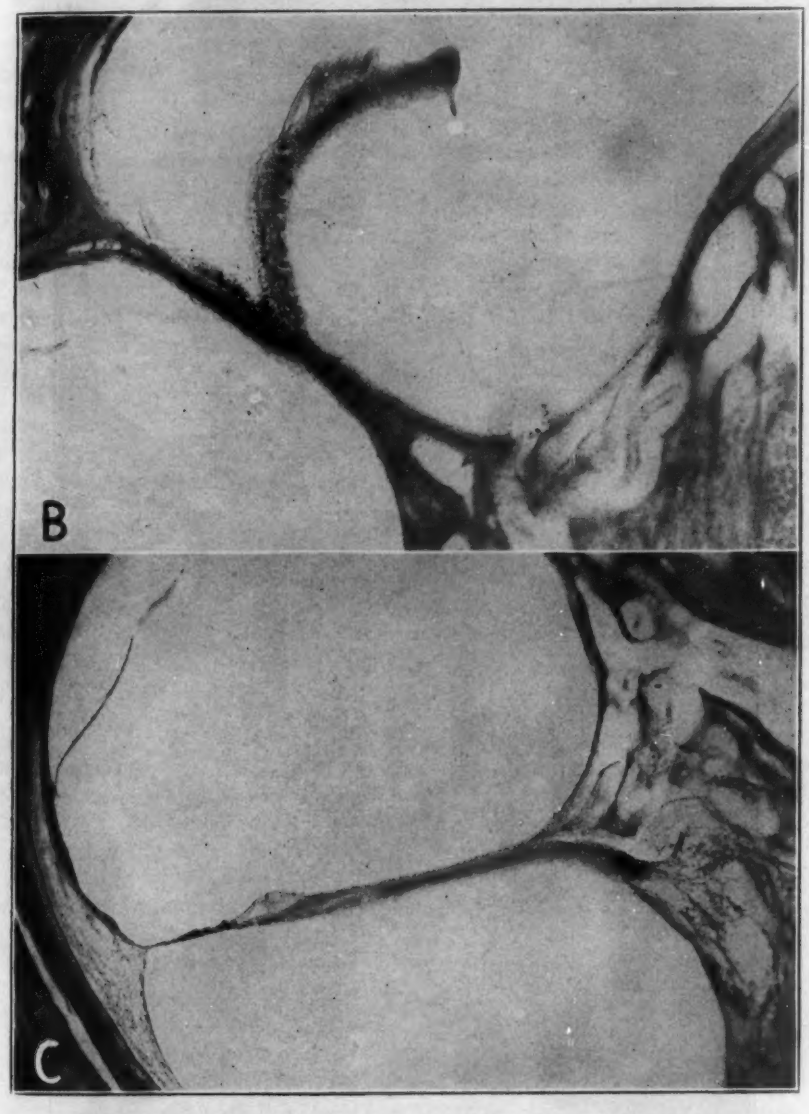

Fig. 4.—*B* and *C*. See legend for *A* on opposite page.

The intact state of the delicate Reissner membrane is interesting in this case and probably is present because the patient, being tensed for the blast, automatically adjusted the stapedius and tensor tympani muscles for protection.

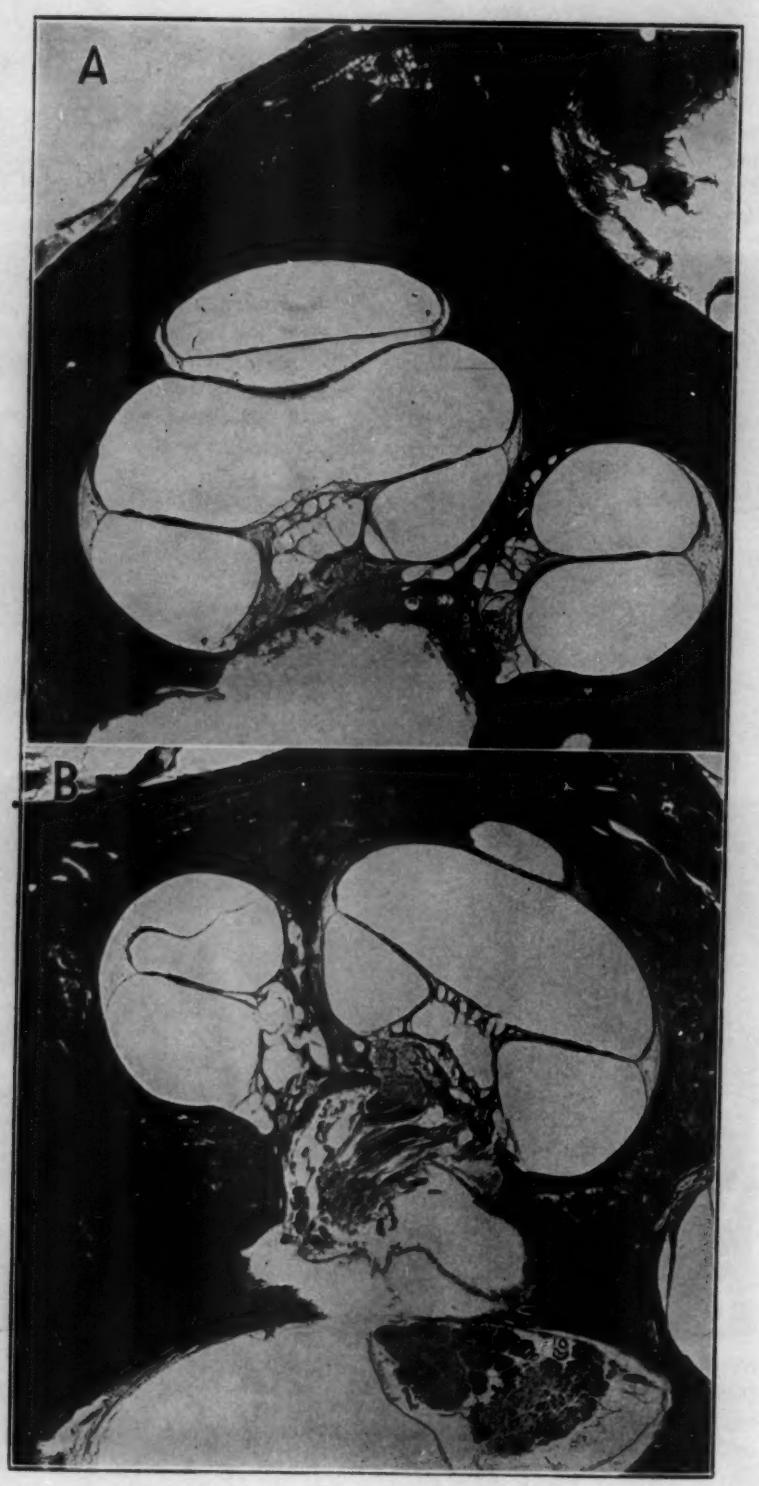

Figure 5

(See legend on opposite page)

CASE 2.—A 52 year old white man, who had worked as a riveter for many years, was hospitalized with massive adenocarcinoma of the stomach and metastatic ascites.

Figure 2 shows his audiometer record (test made by I. Scott, technician). The patient thought his hearing was a "little off." As a child he had little earache and no discharge, true vertigo or tinnitus. The sound in the Weber test was not referred. The Rinne test was positive. Bone conduction was decreased about four seconds. The patient was alert and cooperative. The ward where the test was made was fairly noisy. A notation on the audiometer record said, "The audiometer record shows marked bilateral perceptive deafness with possibly slight bilateral conductive deafness." The external auditory canals were clean; the drum membranes were very thin and transparent. Little if any motion of the handle of the malleus was present. There were no perforations and no inflammation.

Figure 3 illustrates the interesting condition of bilateral fracture of the stapes. On the left in this patient there was a healed fracture of the foot plate of the stapes with callus formation, as illustrated in figure 3 *A*. Figure 3 *B* shows a small fracture of the posterior crus of the right stapes. No callus has formed here, and the bone has failed to knit. On this side the foot plate is dislocated from the anterior joint region but the capsule is still intact. The condition of the inner ear is illustrated in figure 4 *A*, *B* and *C*. In the apical coil the basilar membrane is attached in an abnormal position. Reissner's membrane is torn and floating free throughout the cochlea. The organ of Corti is markedly atrophic throughout, and the spiral ganglion cells are greatly reduced in number.

CASE 3.—The patient was a 46 year old pipe fitter and welder. He occasionally suffered from tinnitus. In the past few years he had also suffered from colds. Five years before examination he had sinus trouble. His ears got "full of dirt" quite often, and he got hard of hearing. The results of the Kahn and Wassermann tests of the blood were doubtful. Repeated Kahn tests were negative. He was in the hospital with rheumatic heart disease, mitral stenosis and cardiac decompensation.

Microscopic examination of the temporal bone fails to reveal Reissner's membrane in either cochlea (fig. 5). The organ of Corti is not normal in any turn. The tectorium also is absent. The spiral ganglion cells are reduced in number. There is evidence of edema in this region. On the left the ganglion cells of the basal turn are extremely angular.

Bilateral centers of otosclerosis are present near the basal turn of the cochleas. These are not in the usual site of predilection, and they do not invade the cochlea. They could not have impaired the hearing (fig. 6).

The eustachian tubes are widely patent to the glandular area, and their condition does not explain the tinnitus. The question arises: Can destruction of the vestibular (Reissner's) membrane produce tinnitus?

COMMENT

Observations on the hearing acuity of persons routinely exposed to loud noises are essential for a proper study of the damage perpetrated. Habermann [1] quoted Hartmann's observation that boilermakers have

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EXPLANATION OF FIGURE 5

A, left ear from a welder and pipe fitter (case 3) who suffered from tinnitus and was "hard of hearing." Note the absence of Reissner's membrane. *B*, right ear.

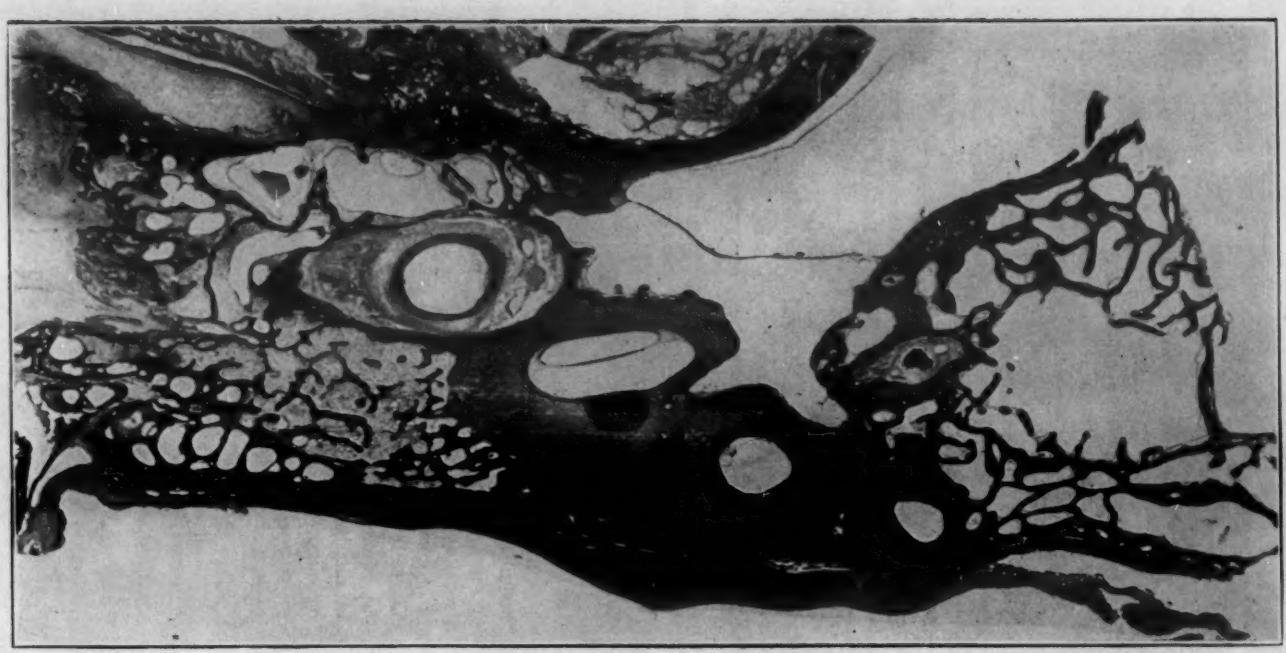

Fig 6 (case 3).—A section through the basal turn of the cochlea in the right ear shown in figure 5*B*. Note the location of the dark-staining otosclerotic center of bone below the cochlea. A similar center occurred in the same relative location on the left.

deafness chiefly for high tones (C_4 and G_4). He also referred to an investigation by Holt of 40 boilermakers, only 6 of whom could hear the spoken voice well. He felt that the difficulty was not in the sound-perceptive but in the sound-conducting apparatus. This he attributed to repeated catarrhal conditions. Habermann [2] himself tested the hearing of 31 boilermakers ranging in age from 18 to 67. He reported specifically the microscopic observations in the cases of 5—1 boilermaker, 2 blacksmiths, 1 artilleryman and 1 machinist. He found atrophy of Corti's organ in most cases in the basilar turn. Corresponding changes occurred in the nerve fibers but seldom in the ganglion cells. The youngest of the 5 workers was 43 years old. The others were in their fifties and sixties. It is now well known that atrophy of the end organ, nerve and ganglion cells occurs with decreased acuity of hearing in the advancing decades of life. Losanow [5] examined the hearing of 54 industrial workers in a nail factory. Apparently most of the workers attempted to protect their ears with cotton. This did not seem to give proper protection. The phonoreceptor organ was injured in from "71-57%." In about 25 per cent of the cases the statoreceptory function was also affected. White [6] reviewed the doubtful probability of damage to the ear in telephone operators. Bunch's [7] observations of a more or less constant type of audiometer record in cases of deafness with a history of occupational trauma are well known.

Some idea of the effects of modern warfare on the human ear as seen in the last World War may be gained from reading Colledge.[8] Reports such as those of Passe [9] and others [10] come in from the present British war zone. Otologists are confronted with a need to know the specific type of lesion produced by specific trauma in order properly to protect those exposed.

Modern warfare increases the possibility of damage to the inner ear of the aviator, the soldier, the iron-worker and the civilian alike. Pro-

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5. Losanow, N.: Professional Traumatization of the Ear of Industrial Workers, Acta oto-laryng. **14**:393-438, 1930.

6. White, J. A.: The Telephone and the Ear, Tr. Am. Otol. Soc. **18**:171-178, 1928.

7. Bunch, C. C.: The Diagnosis of Occupational or Traumatic Deafness: A Historical and Audiometric Study, Laryngoscope **47**:615-691 (Sept.) 1937; Traumatic Deafness from Explosion of a Firecracker: Case Report, Ann. Otol., Rhin. & Laryng. **47**:1092-1095 (Dec.) 1938.

8. Colledge, L.: Injuries to the Ear in Modern Warfare, J. Laryng. & Otol. **36**:283-287, 1921.

9. Passe, E. R. G.: Gunfire Deafness, Brit. M. J. **2**:295 (Aug. 31) 1940.

10. Bomb Concussions and the Ear, Annotations, Lancet **1**:1054 (June 8) 1940. Traumatic Deafness, ibid. **2**:607 (Nov. 15) 1941.

tective plugs for the external canals are suggested by some writers (Dickson and Ewing [11]).

Experimental work along this line on animals was begun in the last war by Guild.[12] Losanow [5] also contributed to the microscopic observations on experimental animals.

Modern animal experimentation has been improved by the development of electrical technic and by the use of conditioned responses. Finch and Culler [13] employed these methods with enlightening results. Horton [14] and Kemp [15] likewise contributed to the present knowledge of the irreparable damage from exposure to continuous noises, through similar methods. Such technic coupled with microscopic studies and reenforced by microscopic studies on human ears known to have undergone traumatic exposure will eventually lead to authentic conclusions.

Washington University School of Medicine.

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11. Dickson, E. D. D., and Ewing, A. W. G.: The Protection of Hearing, J. Laryng. & Otol. **56**:225-242 (July) 1941.

12. Guild, S. R.: War Deafness and Its Prevention: Report of the Animals Used in Testing of Preventive Measures, J. Lab. & Clin. Med. **4**:153-180 (Jan.) 1919.

13. Finch, G., and Culler, E.: Exposure to a Loud Tone, Science **80**:41-42 (July 13) 1934.

14. Horton, G. P.: The Effect of Intense Prolonged Acoustical Stimulations on the Auditory Sensitivity of Guinea Pigs, J. Comp. Psychol. **18**:405-418, 1934; An Experimental Study of Stimulation Deafness in Guinea Pigs, Ann. Otol., Rhin. & Laryng. **44**:252-259 (March) 1935.

15. Kemp, E. H.: An Experimental Investigation of the Problem of Stimulation Deafness, J. Exper. Psychol. **19**:159-171 (April) 1936; A Critical Review of Experiments on the Problem of Stimulation Deafness, Psychol. Bull. **32**:325-342 (May) 1935.

INFLUENCE OF VITAMIN C ON THE ANTI-HISTAMINE ACTION OF VARIOUS DRUGS

EFFECT ON BRONCHIOLAR REACTIONS TO EPHEDRINE, EPINEPHRINE, BENZEDRINE AND CALCIUM, AS STUDIED BY MICROSCOPIC OBSERVATION

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The use of experimental methods in establishing the physiologic properties of the therapeutic agents commonly employed in the respiratory tract has been given an effective impetus through the work of Proetz[1] on ciliary action of the nasal mucosa. The microscopic observation of bronchiolar reactions opens up an additional experimental method for evaluation of the effect of drugs on the lower respiratory tract.

Outstanding among the problems of rhinology is that of the circulation. The constant play between vasodilatation and vasoconstriction directs a large part of present therapeutic efforts. This is particularly important for the drugs that are commonly used in the upper respiratory tract, such as ephedrine, epinephrine, benzedrine and calcium.

Do these drugs which constrict the nasal mucosa increase or diminish bronchiolar size? Are the various salts of these compounds of equal activity, and what effect, if any, does vitamin C have on them? These were some of the questions on which experimental data were desirable.

In histamine one has a drug that acts as a vasodilator of the arterioles and capillaries and constricts the bronchioles, producing the typical picture of anaphylaxis; in anaphylactic shock histamine is liberated from the liver of the dog and from the lung of the guinea pig. The histamine effect has frequently been suggested as an underlying factor in allergic sensitivity and surgical shock. For experimental purposes the neutralization of the histamine effect by epinephrine, benzedrine, ephedrine or calcium can be evaluated by microscopic observation of the bronchiolar reaction of the rabbit lung.

1. Proetz, A.: Essays on the Applied Physiology of the Nose, St. Louis, Annals Publishing Company, 1941.

METHOD OF INVESTIGATION

For the purpose of this study the vitamin C salts of the aforementioned compounds were compared with the commonly used preparations. In this manner a most interesting synergistic action was revealed for all four preparations.

The object of the experiment was to secure a viable section of bronchiole. This was accomplished by fixing the lung of the rabbit in gelatin, chilling it and then cutting a thin section of a bronchiole, which was kept in Ringer-Locke dextrose solution. The section was mounted on a ring so that when the gelatin was dissolved out the reactions of the bronchiole could be observed under the microscope and drawn to size. The whole procedure was conducted with adequate controls to assure the viability of the bronchiolar sections.

I first observed the technic of the microscopic study of bronchiolar reactions in the laboratory of Dr. H. D. Pease, who had constructed an ingenious microscope platform that permitted maintenance of a water bath of constant temperature for the ordinary Petri dish. This could readily be elaborated for a series of microscopes. In general, the technic of Sollmann and Gilbert[2] was used as follows: Each of the following substances was dissolved separately in 500 cc. of water:

	Gm.
Sodium chloride	31.5
Potassium chloride	1.47
Calcium chloride	0.84
Sodium bicarbonate ($NaHCO_3$)	1.15

The solutions were mixed and diluted to 3.5 liters. On the day of use, dextrose, 1 Gm. per liter, was added. Substances to be tested which are sufficiently soluble that a dilution of 1: 50 is adequate are made up in distilled water. Those which are less soluble are made up in Ringer-Locke solution.

The animal was killed by intravenous injection of air. The skin was removed from the ventral portion of the abdomen, the thorax and the neck. A midline incision was made from the middle of the abdomen to the diaphragm. Lateral incisions were made just posterior to the diaphragm toward the sides of the body.

The diaphragm was punctured on each side, allowing the lungs to collapse. The ventral thoracic wall was removed, longitudinal cuts being made along the sides of the thorax anterior to the first pair of ribs. Care was taken not to cut the large axillary veins. A hemostat should be used on any small vessels which are accidentally cut. The first rib and the episternum were carefully removed, carrying with them the ventral neck muscles. The trachea was cut and a cannula inserted, which was held in place with hemostats. The lungs were filled to approximately normal expansion with gelatin (approximately 15 cc. of warm 10 per cent gelatin dissolved in Ringer-Locke solution for each kilogram of body weight). The inferior vena cava was clamped with a hemostat, and the lungs, heart and trachea were removed and placed in ice-cold Ringer-Locke solution (approximately 150 cc.). The organs were placed in the freezing compartment of the refrigerator for one to one and a half hours.

The two large lobes of the lung were trimmed to approximately one-half size and thin sections of tissue cut across the bronchiole. These sections should be

2. Sollmann, T., and Gilbert, A. J.: Microscopic Observations of Bronchiolar Reactions, J. Pharmacol. & Exper. Therap. **61**:272, 1937.

as thin as possible (0.5 to 1 mm. in thickness). The sections were placed in the cold Ringer-Locke solution in which the lungs were kept. The sections were kept ice cold until each one was used.

If the substance to be tested was to be diluted, 49 cc. of Ringer-Locke solution was placed in the special dishes (9 cm. Petri dishes, in the center of which a ring of cork with a central opening, approximately 5 cm. in diameter, was cemented with beeswax). The section to be used was pinned out with the bronchiole over the opening of the cork. The sections should not be stretched but should be pinned out flat. The dish was placed on the warm stage and the contents of the dish allowed to warm to 37 C., the section being agitated by means of a stream of Ringer-Locke solution from a small pipet. Camera lucida drawings of the bronchiolar lumen were made until it was of constant size (this usually took ten to fifteen minutes). The test solution was added near the edge of the Petri dish and mixed with the pipet. Then a small amount of fluid in the dish was picked up with the pipet and allowed to run out gently over the tissue. Camera lucida drawings were made as indicated by the reaction of the bronchiole.

In tests designed to show possible antagonism between two substances it was found that the second substance should generally be added about five minutes after the first. The drawings made for each test were dated and a number assigned to each. Each drawing was labeled with the exact time of day, and later these times were translated into terms of minutes which elapsed after the section was put into the dish. These drawings were filed as a permanent record.

Computation of Results.—The area of the bronchiolar lumen was measured in arbitrary units by means of a polar planimeter. The area just before the addition of the first substance to be tested was taken as 100 per cent, and was called the original area. Reactions were expressed as percentages of this original area which remained.

The animals used were white New Zealand rabbits, partially standardized as to age, weight, health and environment. Five animals were prepared and used as described.

The concentration of the solutions was kept equal for all comparative tests. The solution of histamine was made up with 100 mg. of histamine hydrochloride to 100 cc. of Ringer-Locke solution. The dilution factor for all solutions of test substances was 1 : 50, giving with the histamine solution a 1 : 50,000 concentration.

Experiments 1 and 2 for each day were made as controls. The experiments were arranged in the order of the days on which they were conducted so that the daily controls could be checked against the day's experiments. For each of five groups a new animal was killed, and the experiments and the controls were run simultaneously.

The purpose of the experiment was to produce contraction of the bronchiole by adding histamine hydrochloride to the Ringer-Locke-dextrose solution to obtain a 1 : 50,000 concentration. After the contraction due to the histamine was established, and at a fixed interval, the substance to be tested for histamine antagonism was added. Those substances which antagonize histamine allowed the bronchiole to relax and in some instances continued the dilatation above normal. The

control tests also showed that there was a spontaneous tendency for the histamine effect to diminish, with a secondary histamine contraction after about fifteen minutes. As a countercheck for histamine antagonism, therefore, two sets of tests were run. In one series, the substance to be tested was added after the histamine contraction had occurred, and the effect was noted. In the other, histamine contraction was produced, the substance added and the histamine antagonism noted; then histamine was again added to see whether the histamine antagonism of the substance would prevent further histamine effect. With substances having histamine-antagonistic properties, then, there would be no or little contraction of the bronchiole on the secondary addition of histamine. The controls uniformly showed a secondary contraction when the histamine was added for the second time. This double checking system thus provided useful information.

Among the many problems besetting the use of various pharmacologic agents in rhinologic practice is the question whether there is any difference in pharmacologic action between the various salts of the alkaloids or metals. Does the hydrochloride act like the sulfate? Do the vitamins have a relationship to the other therapeutic agents? Vitamin C (ascorbic acid) has been claimed to bear an important relationship to the adrenal gland and to epinephrine. It is here demonstrated to have a synergistic effect on calcium. For these and other questions an experimental approach is available in the bronchiolar reactions. The experiments are arranged in the order of the days on which they were conducted so that the daily controls can be checked against the day's experiments.

RESULTS OF INVESTIGATION

GROUP 1.—Experiments 1 and 2: These tests were conducted to establish normal as well as histamine controls and a subsequent histamine control after twenty-four minutes. Results for 6 normal and 6 histamine controls were plotted and normal and histamine curves established.

On the day of these experiments the normal control maintained a fairly steady bronchiolar lumen, with only gradual contraction. The histamine control showed rapid contraction of the bronchiole with recovery and a second contraction under the continued action of the histamine, thus causing a secondary drop after seventeen minutes.

Experiments 3 and 4: These tests were conducted with the same tissues as those used in experiments 1 and 2. When sodium ascorbate was added after the histamine recovery was slower than in the control, but the histamine antagonism was evident on the addition of the second dose of histamine hydrochloride after fourteen minutes. There was no secondary histamine contraction.

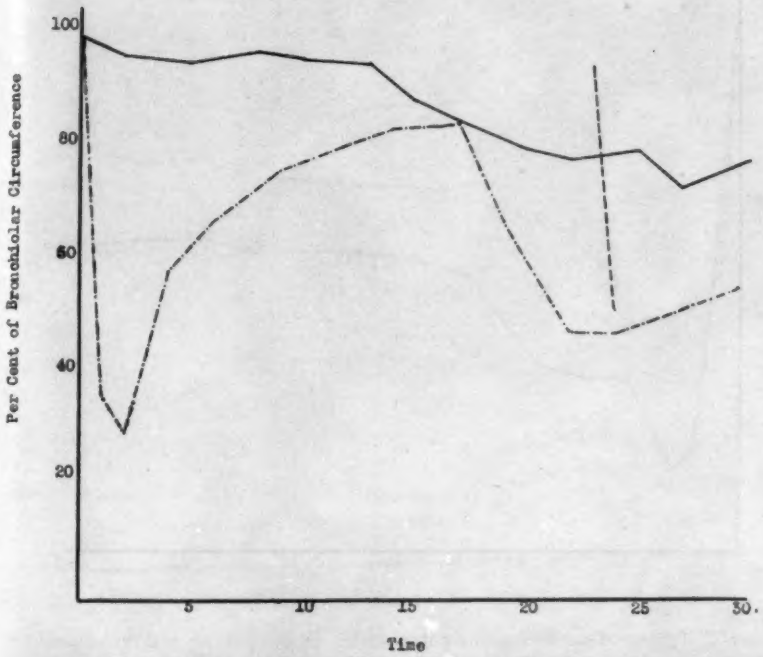

Chart 1 (group 1).—Effect of histamine hydrochloride on bronchiolar lumen. Experiment 1: Normal control (solid line). Experiment 2: Histamine control, zero minutes (line of dots and dashes); subsequent histamine control, twenty-four minutes (line of dashes). In this chart and in the accompanying charts, the number of minutes refers to the time at which the drug was added to the Ringer-Locke solution.

TABLE 1.—*Bronchiolar Response to Histamine Hydrochloride (Control Experiments)*

Test Substance	Final Concentration of Test Substance	Time of Addition (Min.)	Time of Observation (Min.)	Area, Arbitrary Units	Percentage of Original Area
Group 1		Experiment 1			
Normal control.....	.....	0	0	5.78	100.0
			2	5.61	97.0
			5	5.52	95.5
			8	5.62	97.2
			10	5.55	96.0
			13	5.50	95.1
			15	5.11	88.4
			17	4.92	85.1
			20	4.62	79.9
			22	4.51	78.0
			25	4.60	79.5
			27	4.23	73.1
			30	4.50	77.8
		Experiment 2			
Histamine hydrochloride..	1:50,000	0	0	5.60	100.0
			1	2.03	36.2
			2	1.68	30.0
			4	3.26	58.2
			6	3.76	67.1
			9	4.26	76.0
			12	4.52	80.7
			14	4.68	83.5
			17	4.71	84.1
			19	3.76	67.1
			22	2.63	47.5
			24	2.65	47.3
			27	2.90	51.7
			29	3.09	55.1

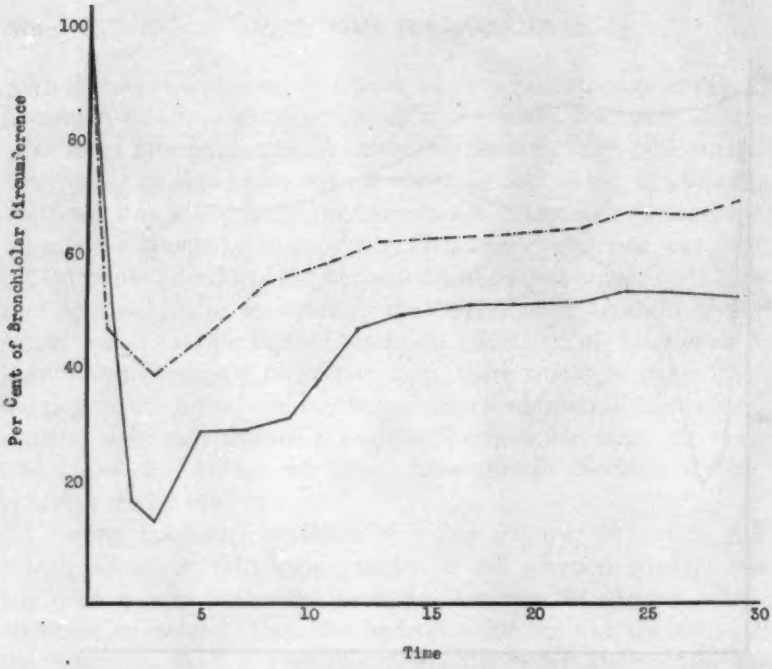

Chart 2 (group 1).—Histamine-antagonistic properties of sodium ascorbate. Experiment 3: Histamine hydrochloride, zero minutes; sodium ascorbate, four minutes (solid line). Experiment 4: Histamine hydrochloride, zero minutes; sodium ascorbate, four minutes; histamine hydrochloride, fourteen minutes (line of dots and dashes).

TABLE 2.—*Antihistamine Action of Sodium Ascorbate*

Test Substance	Final Concentration of Test Substance	Time of Addition (Min.)	Time of Observation (Min.)	Area, Arbitrary Units	Percentage of Original Area
Group 1		Experiment 3			
Histamine hydrochloride..	1:50,000	0	0	3.21	100.0
			2	0.59	18.3
			3	0.47	14.6
Sodium ascorbate.....	1:750	4	5	0.97	30.2
			7	1.00	31.1
			9	1.07	33.3
			12	1.57	48.9
			14	1.66	51.7
			17	1.71	53.2
			19	1.71	53.2
			22	1.72	53.5
			24	1.79	55.7
			26	1.77	55.1
			29	1.75	54.5
		Experiment 4			
Histamine hydrochloride..	1:50,000	0	0	2.92	100.0
			1	1.41	48.2
			3	1.18	40.4
Sodium ascorbate.....	1:750	4	5	1.38	47.2
			8	1.67	57.1
			11	1.78	60.9
			13	1.67	64.0
Histamine hydrochloride..	1:50,000	14	15	1.89	64.7
			19	1.92	65.7
			22	1.94	66.4
			24	2.04	68.7
			27	2.02	69.1
			29	2.09	71.5

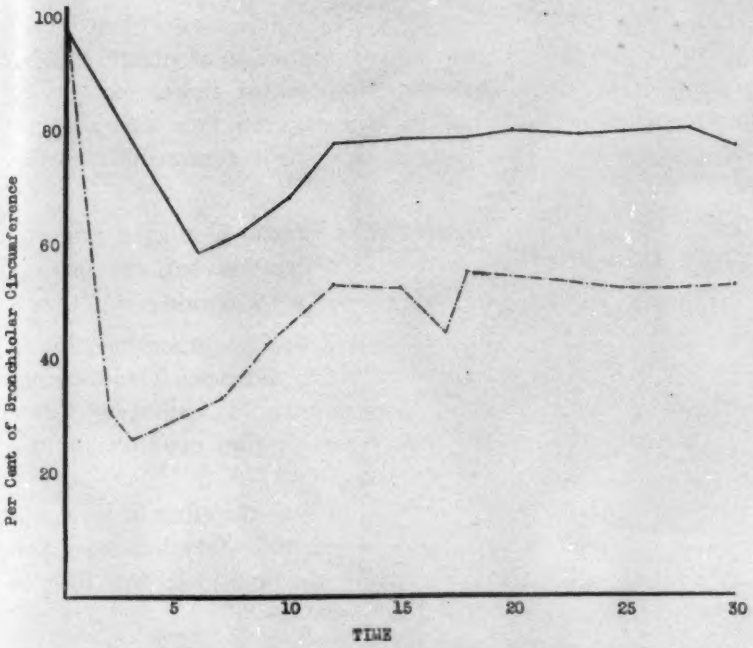

Chart 3 (group 1).—Histamine-antagonistic properties of vitamin C. Experiment 5: Histamine hydrochloride, five minutes; vitamin C (ascorbic acid), nine minutes (solid line). Experiment 6: Histamine hydrochloride, one minute; vitamin C, five minutes; histamine hydrochloride, sixteen minutes (line of dots and dashes).

TABLE 3.—*Antihistamine Effect of Vitamin C (Ascorbic Acid)*

Test Substance	Final Concentration of Test Substance	Time of Addition (Min.)	Time of Observation (Min.)	Area, Arbitrary Units	Percentage of Original Area
Group 1		Experiment 5			
Histamine hydrochloride..	1:50,000	5	0	5.35	100.0
			6	3.23	60.3
			8	3.46	64.6
Vitamin C.....	1:750	9	10	3.77	70.4
			15	4.31	80.5
			18	4.34	81.1
			20	4.40	82.2
			23	4.37	81.6
			25	4.39	82.0
			28	4.44	82.9
			30	4.25	79.4
		Experiment 6			
Histamine hydrochloride..	1:50,000	1	0	3.75	100.0
			2	1.23	32.8
			8	1.05	28.0
Vitamin C.....	1:750	5	7	1.31	34.9
			9	1.66	44.2
			12	2.08	55.4
			15	2.05	54.6
Histamine hydrochloride..	1:50,000	16	17	1.75	46.6
			18	2.17	57.8
			22	2.10	56.0
			25	2.05	54.6
			27	2.04	54.4
			30	2.06	54.9

Experiments 5 and 6: The histamine antagonism of vitamin C alone was evident. Here, again, there was a somewhat slower recovery of the bronchiolar lumen, and recovery was maintained, even after a second dose of histamine, although there was a slight contraction for two minutes.

Summary.—The sodium ascorbate and vitamin C did not influence the speed of recovery from histamine contraction but did produce antagonism to the second dose of histamine hydrochloride.

GROUP 2.—On this day controls showed that the bronchiolar lumen maintained its normal size for the whole thirty minutes. The histamine response was strongly active after thirty minutes, indicating that a secondary histamine contraction was obtainable after exposure for this period.

Experiments 3 and 4: Normal recovery from the effect of histamine hydrochloride was slightly inhibited by ephedrine sulfate, indicating that the drug had a slight constricting action on the bronchiole, with little or no antagonism to the second dose of histamine.

Experiments 5 and 6: Ephedrine ascorbate produced prompt recovery from a sharper histamine contraction than that showed in the control, with maintenance of a good level of histamine antagonism after the second dose.

Experiments 7 and 8: With ephedrine hydrochloride there was a decidedly increased histamine contraction as compared with the response with either ephedrine ascorbate or ephedrine sulfate, as well as delayed recovery. Ephedrine hydrochloride is therefore a bronchiole-constricting agent with no histamine antagonism.

Summary.—Ephedrine sulfate caused slight inhibition of recovery from histamine hydrochloride, with little or no antagonism to the second dose of histamine. With ephedrine ascorbate there were quick recovery from histamine and fair antagonism to the second dose of histamine. With ephedrine hydrochloride there was an increase in histamine contraction and no histamine antagonism, rather constriction.

GROUP 3.—Controls for this day showed normal maintenance of the bronchiolar lumen for thirty minutes and an active secondary response to histamine hydrochloride after thirty minutes.

Experiments 3 and 4: Experiments conducted with calcium ascorbate showed a sharp recovery after the first histamine contraction, followed by a moderate drop and a moderate rise after the second dose of histamine. This was more rapid than the response with either vitamin C alone or sodium ascorbate.

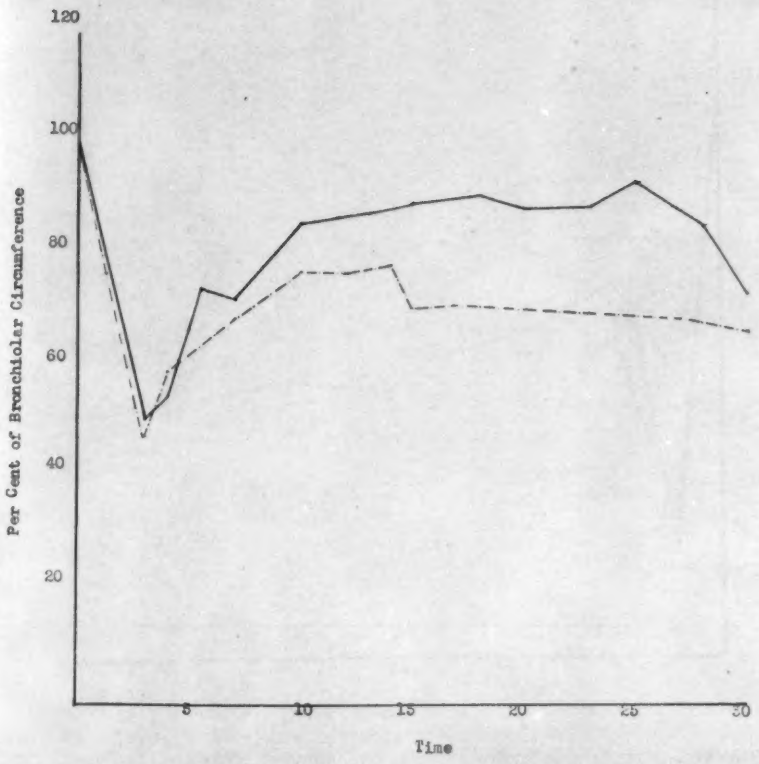

Chart 4 (group 2).—Histamine-antagonistic action of ephedrine sulfate. Experiment 3: Histamine hydrochloride, two minutes; ephedrine sulfate, five minutes (solid line). Experiment 4: Histamine hydrochloride, one minute; ephedrine sulfate, three and a half minutes; histamine hydrochloride, thirteen minutes (line of dots and dashes).

TABLE 4.—*Antihistamine Effect of Ephedrine Sulfate*

Test Substance	Final Concentration of Test Substance	Time of Addition (Min.)	Time of Observation (Min.)	Area, Arbitrary Units	Percentage of Original Area
Group 2		Experiment 3			
		..	0	5.35	100.0
Histamine hydrochloride..	1:50,000	2	3	2.72	50.8
			4	2.92	54.5
Ephedrine sulfate.....	1:10,000	5	5½	3.96	74.0
			7	3.86	72.1
			10	4.58	85.6
			13	4.66	87.1
			15	4.76	88.9
			18	4.85	90.6
			20	4.72	88.2
			23	4.77	89.1
			25	4.98	93.0
			28	4.58	85.4
			30	3.93	73.4
		Experiment 4			
Histamine hydrochloride..	1:50,000	..	0	5.21	100.0
		1	2	2.22	42.6
			3	2.50	47.9
Ephedrine sulfate.....	1:10,000	3½	4	3.08	59.1
			7	3.56	68.3
			10	4.01	76.9
			12	4.00	76.7
Histamine hydrochloride..	1:50,000	13	14	4.08	78.3
			15	3.68	70.6
			17	3.71	71.2
			27	3.50	68.9
			30	3.47	66.6

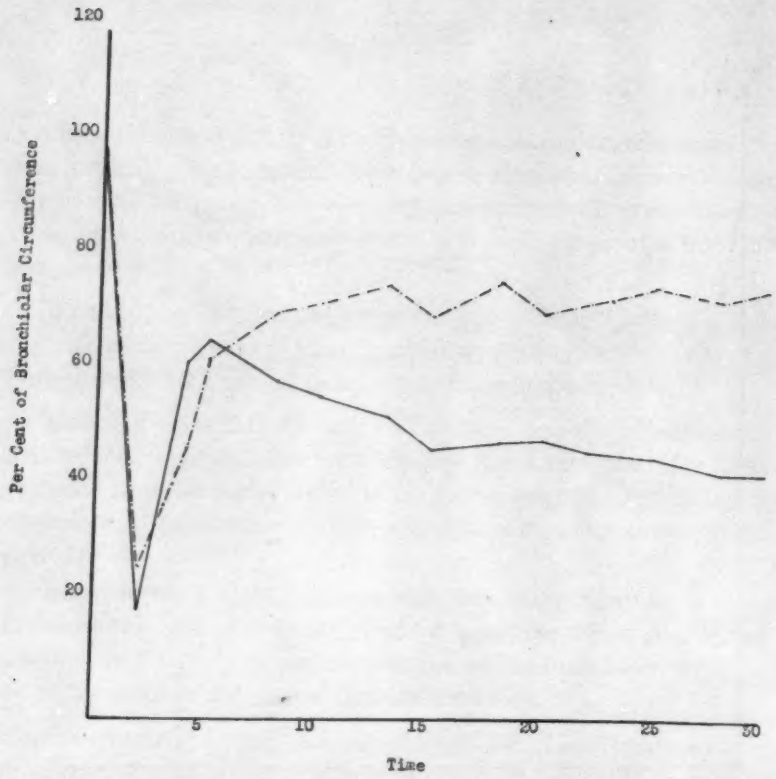

Chart 5 (group 2).—Histamine-antagonistic action of ephedrine ascorbate. Experiment 5: Histamine hydrochloride, one minute; ephedrine ascorbate, three minute (solid line). Experiment 6: Histamine hydrochloride, one minute; ephedrine ascorbate, three minutes; histamine hydrochloride, fourteen minutes (line of dots and dashes).

TABLE 5.—*Antihistamine Effect of Ephedrine Ascorbate*

Test Substance	Final Concentration of Test Substance	Time of Addition (Min.)	Time of Observation (Min.)	Area, Arbitrary Units	Percentage of Original Area
Group 2		Experiment 5			
		..	0	2.67	100.0
Histamine hydrochloride..	1:50,000	1	2	52	19.4
Ephedrine ascorbate.....	1:10,000	3	4	1.67	62.5
			5	1.78	66.6
			8	1.60	59.9
			10	1.52	56.9
			13	1.43	53.5
			15	1.28	47.9
			18	1.31	49.0
			20	1.32	49.4
			22	1.27	47.5
			25	1.24	46.4
			28	1.17	43.8
			30	1.17	43.8
		Experiment 6			
		..	0	0.57	100.0
Histamine hydrochloride..	1:50,000	1	2	1.75	26.6
Ephedrine ascorbate.....	1:10,000	3	4	3.11	47.3
			5	4.16	63.3
			8	4.71	71.7
			10	4.84	73.6
			13	5.05	76.8
Histamine hydrochloride..	1:50,000	14	15	4.70	71.5
			18	5.11	77.7
			20	4.73	71.9
			23	4.92	74.8
			25	5.06	77.0
			28	4.88	74.2
			30	5.00	76.1

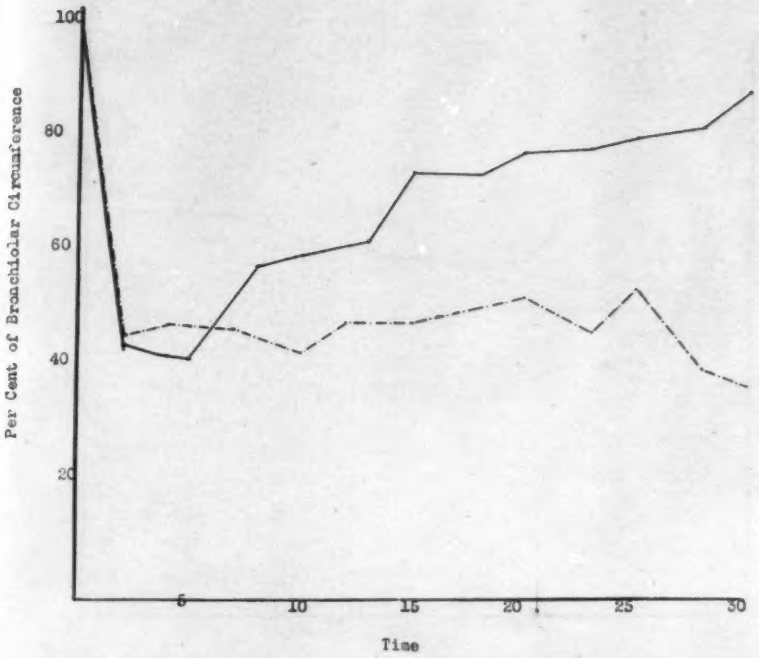

Chart 6 (group 2).—Histamine-antagonistic action of ephedrine hydrochloride. Experiment 7: Histamine hydrochloride, one minute; ephedrine hydrochloride, three minutes (solid line). Experiment 8: histamine hydrochloride, one minute; ephedrine hydrochloride, three minutes; histamine hydrochloride, fourteen minutes (line of dots and dashes).

TABLE 6.—*Antihistamine Effect of Ephedrine Hydrochloride*

Test Substance	Final Concentration of Test Substance	Time of Addition (Min.)	Time of Observation (Min.)	Area, Arbitrary Units	Percentage of Original Area
Group 2		Experiment 7			
		..	0	5.13	100.0
Histamine hydrochloride..	1:50,000	1	2	2.30	44.8
Ephedrine hydrochloride..	1:10,000	3	4	2.20	42.8
			5	2.18	42.4
			8	3.01	58.6
			10	3.09	60.2
			13	3.25	63.3
			15	3.87	75.4
			18	3.85	75.0
			20	4.04	78.7
			23	4.08	79.5
			25	4.32	81.2
			28	4.27	83.2
			30	4.60	89.6
		Experiment 8			
		..	0	5.36	100.0
Histamine hydrochloride..	1:50,000	1	2	2.48	46.2
Ephedrine hydrochloride..	1:10,000	3	4	2.58	48.1
			7	2.56	47.7
			10	2.35	43.8
			12	2.62	48.8
Histamine hydrochloride..	1:50,000	14	15	2.62	48.8
			17	2.72	50.7
			20	2.85	53.1
			23	2.54	47.3
			25	2.93	54.6
			28	2.15	40.1
			30	2.01	37.5

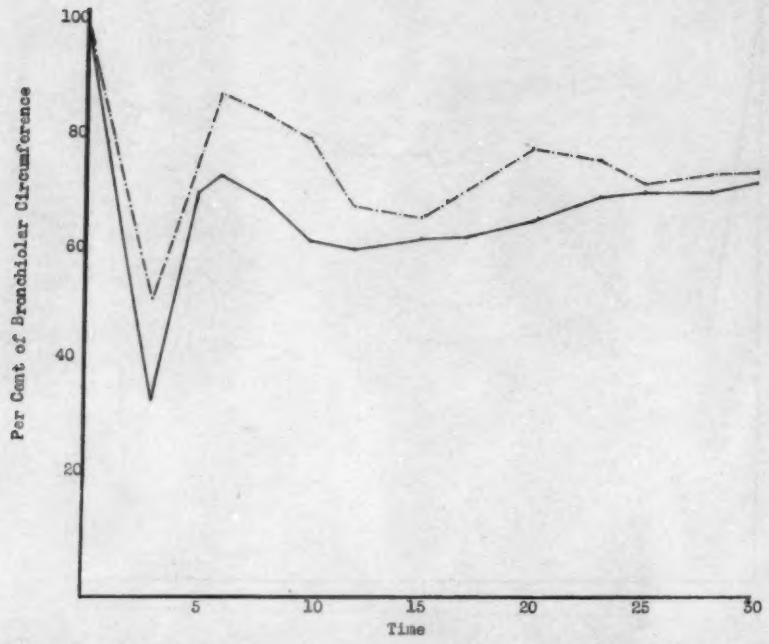

Chart 7 (group 3).—Histamine-antagonistic action of calcium ascorbate. Experiment 3: Histamine hydrochloride, one minute; calcium ascorbate, four minutes (solid line). Experiment 4: Histamine hydrochloride, one minute; calcium ascorbate, four minutes; histamine hydrochloride, eleven minutes.

TABLE 7.—*Antihistamine Effect of Calcium Ascorbate*

Test Substance	Final Concentration of Test Substance	Time of Addition (Min.)	Time of Observation (Min.)	Area, Arbitrary Units	Percentage of Original Area
Group 3		Experiment 3			
		..	0	5.50	100.0
Histamine hydrochloride..	1:50,000	1	3	1.91	34.7
Calcium ascorbate.....	1:750	4	5	3.91	71.0
			6	4.07	74.0
			8	3.85	70.0
			10	3.44	62.5
			12	3.36	61.0
			15	3.48	63.2
			17	3.49	63.4
			20	3.64	66.1
			23	3.88	70.5
			25	3.92	71.2
			28	3.95	71.8
			30	4.01	72.9
		Experiment 4			
		..	0	5.56	100.0
Histamine hydrochloride..	1:50,000	1	3	2.91	52.3
Calcium ascorbate.....	1:750	4	6	4.92	88.4
			8	4.74	85.2
			10	4.47	80.3
Histamine hydrochloride..	1:50,000	11	12	3.82	68.7
			15	3.73	67.0
			20	4.38	78.7
			23	4.29	77.1
			25	4.06	73.0
			28	4.14	74.4
			30	4.17	75.0

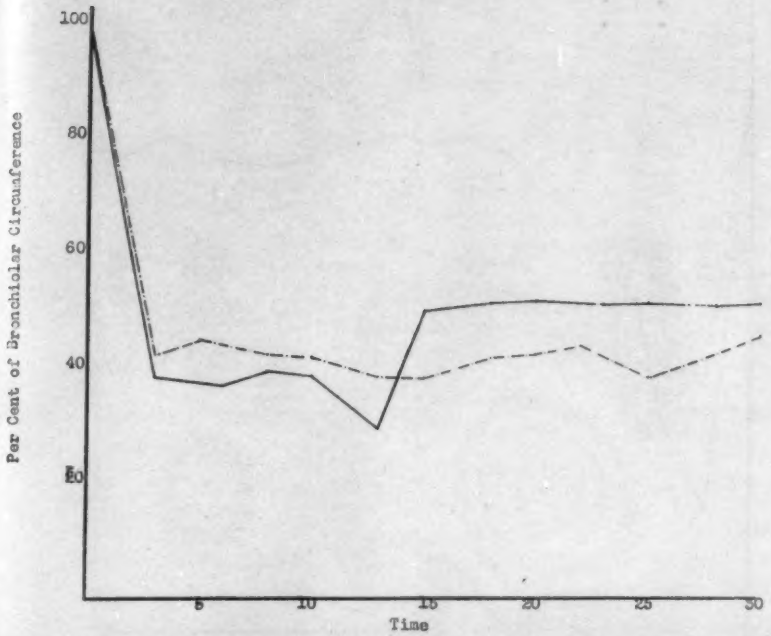

Chart 8 (group 3).—Histamine-antagonistic action of calcium gluconate. Experiment 5: Histamine hydrochloride, one minute; calcium gluconate, four minutes (solid line). Experiment 6: Histamine hydrochloride, one minute; calcium gluconate, four minutes; histamine hydrochloride, eleven minutes (line of dots and dashes).

TABLE 8.—*Antihistamine Effect of Calcium Gluconate*

Test Substance	Final Concentration of Test Substance	Time of Addition (Min.)	Time of Observation (Min.)	Area, Arbitrary Units	Percentage of Original Area
Group 3		Experiment 5			
		..	0	6.95	100.0
Histamine hydrochloride..	1:50,000	1	3	2.73	39.2
Calcium gluconate.....	1:1,000	4	6	2.62	37.9
			8	2.79	40.1
			10	2.76	39.7
			13	2.12	30.5
			15	3.54	50.9
			18	3.65	52.5
			20	3.68	52.9
			23	3.63	52.2
			25	3.65	52.5
			28	3.62	52.0
			30	3.63	52.2
		Experiment 6			
		..	0	4.16	100.0
Histamine hydrochloride..	1:50,000	1	3	1.79	43.0
Calcium gluconate.....	1:1,000	4	5	1.91	45.9
			6	1.80	43.2
			10	1.78	42.7
Histamine hydrochloride..	1:50,000	11	13	1.65	39.6
			15	1.65	39.6
			18	1.79	43.0
			20	1.81	43.5
			22	1.87	44.9
			25	1.66	40.9
			28	1.83	43.9
			30	1.95	46.8

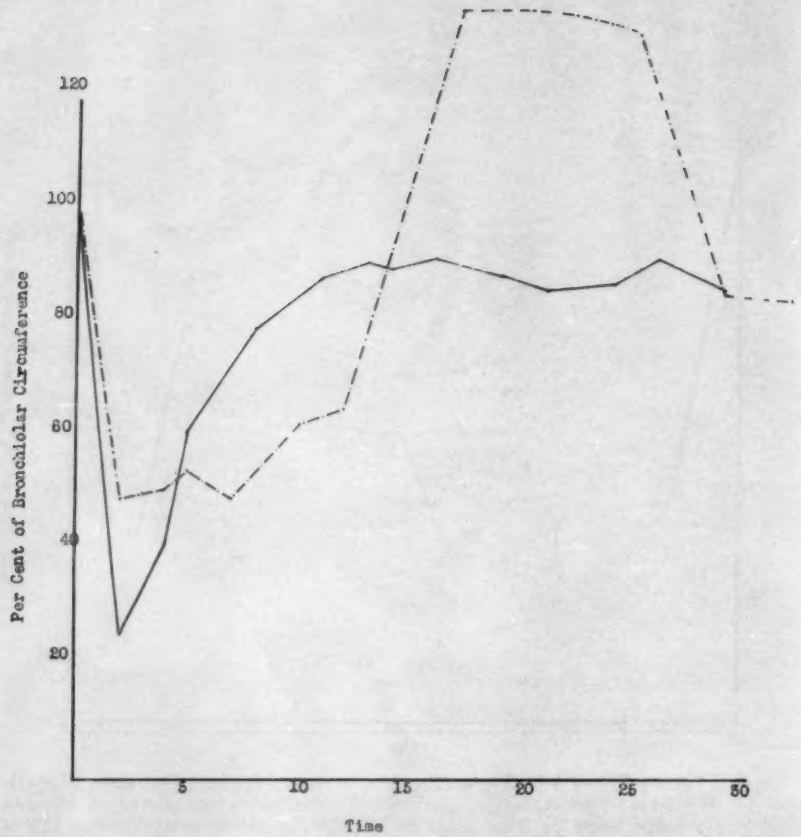

Chart 9 (group 4).—Histamine-antagonistic action of benzedrine ascorbate. Experiment 3: Histamine hydrochloride, one minute; benzedrine ascorbate, three minutes (solid line). Experiment 4: Histamine hydrochloride, one minute; benzedrine ascorbate, three and a half minutes; histamine hydrochloride, nineteen minutes (line of dots and dashes).

TABLE 9.—*Antihistamine Effect of Benzedrine Ascorbate*

Test Substance	Final Concentration of Test Substance	Time of Addition (Min.)	Time of Observation (Min.)	Area, Arbitrary Units	Percentage of Original Area
Group 4		Experiment 3			
		..	0	3.36	100.0
Histamine hydrochloride..	1:50,000	1	2	0.87	25.8
Benzedrine ascorbate.....	1:10,000	3	4	1.40	41.6
			2	2.05	61.0
			8	2.67	79.4
			11	2.97	88.3
			13	3.07	91.3
			14	3.04	90.4
			16	3.10	92.2
			19	3.01	89.5
			21	2.92	86.9
			24	2.96	88.0
			26	3.11	92.5
			29	2.93	87.2
		Experiment 4			
		..	0	1.77	100.0
Histamine hydrochloride..	1:50,000	1	2	0.88	49.7
			3	0.89	50.2
Benzedrine ascorbate.....	1:1,000	3½	4	0.90	50.8
			5	0.96	54.2
			7	0.88	49.7
			10	1.11	62.7
			12	1.16	65.5
			17	2.42	136.7
Histamine hydrochloride..	1:50,000	19	20	2.42	136.7
			22	2.41	136.1
			25	2.37	133.8
			29	1.53	86.4
			32	1.52	85.5

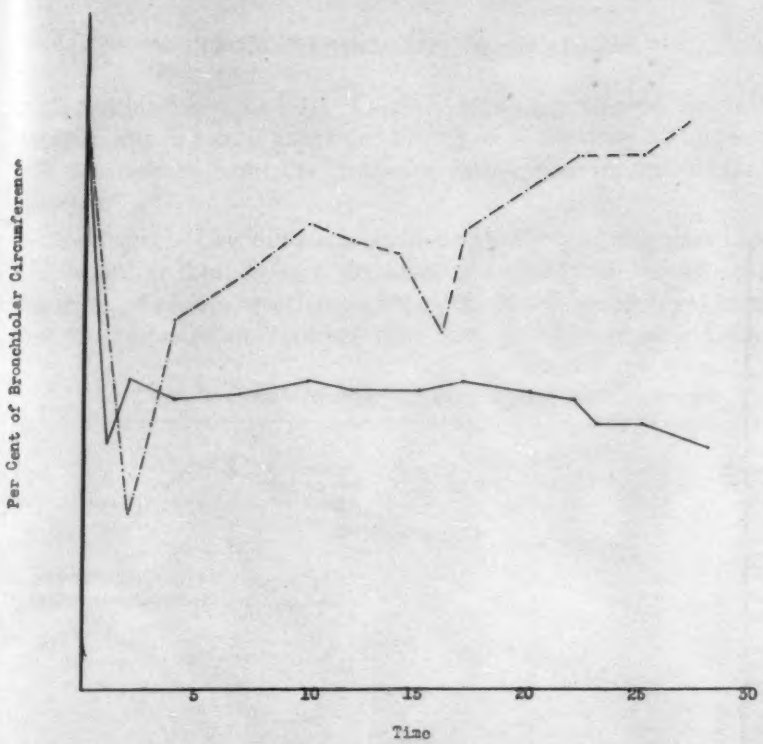

Chart 10 (group 4).—Histamine-antagonistic action of benzedrine sulfate. Experiment 5: Histamine hydrochloride, one-half minute; benzedrine sulfate, one and a half minutes (solid line). Experiment 6: Histamine hydrochloride, one minute; benzedrine sulfate, two and a half minutes; histamine hydrochloride, thirteen minutes (line of dots and dashes).

TABLE 10.—*Antihistamine Effect of Benzedrine Sulfate*

Test Substance	Final Concentration of Test Substance	Time of Addition (Min.)	Time of Observation (Min.)	Area, Arbitrary Units	Percentage of Original Area
Group 4		Experiment 5			
		..	0	2.36	100.0
Histamine hydrochloride..	1:50,000	½	1	1.03	43.6
Benzedrine sulfate.....	1:10,000	1½	2	1.29	54.6
			4	1.22	51.6
			7	1.23	52.1
			10	1.29	54.6
			12	1.26	53.3
			15	1.26	53.3
			17	1.29	54.6
			20	1.25	52.9
			22	1.22	51.6
			23	1.11	47.0
			25	1.11	47.0
			28	1.01	42.7
		Experiment 6			
		..	0	3.45	100.0
Histamine hydrochloride..	1:50,000	1	2	1.07	31.0
Benzedrine sulfate.....	1:10,000	2½	3	1.62	46.9
			4	2.26	65.5
			7	2.54	73.6
			10	2.86	82.8
			12	2.75	79.7
Histamine hydrochloride..	1:50,000	13	14	2.68	77.6
			16	2.19	63.4
			17	2.82	81.7
			22	3.27	94.7
			25	3.27	94.7
			27	3.46	100.2

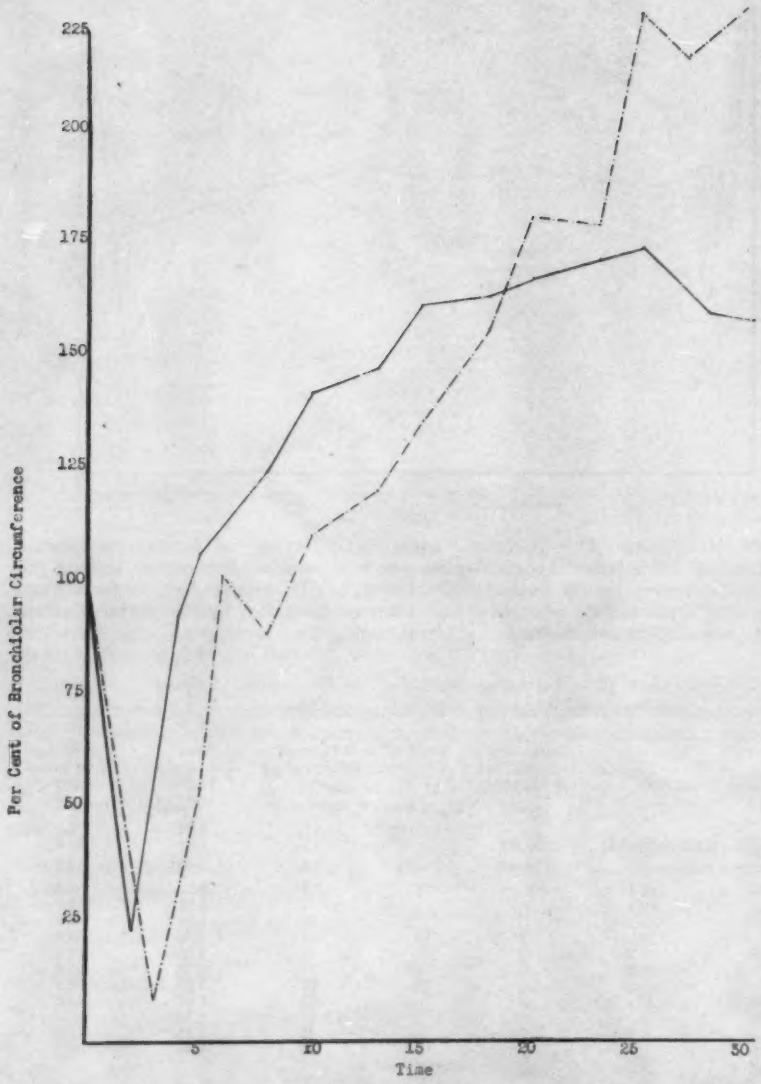

Chart 11 (group 5).—Histamine-antagonistic action of epinephrine ascorbate. Experiment 3: Histamine hydrochloride, one minute; epinephrine ascorbate, three minutes (solid line). Experiment 4: Histamine hydrochloride, two minutes; epinephrine ascorbate, four minutes; histamine hydrochloride, fourteen minutes (line of dots and dashes).

Experiments 5 and 6: Calcium gluconate showed no histamine antagonism; in fact, it prolonged the action of the drug. The bronchiole did not recover from the histamine contraction for the whole thirty minutes.

Summary.—Calcium gluconate not only did not antagonize histamine hydrochloride, but, in fact, increased contraction of the bronchiole to the drug. Calcium ascorbate, on the other hand, antagonized histamine, and the response was quicker than that to either vitamin C alone or

TABLE 11.—*Antihistamine Effect of Epinephrine Ascorbate*

Test Substance	Final Concentration of Test Substance	Time of Addition (Min.)	Time of Observation (Min.)	Area, Arbitrary Units	Percentage of Original Area
Group 5		Experiment 3			
		..	0	3.19	100.0
Histamine hydrochloride..	1:50,000	1	2	0.80	25.0
Epinephrine ascorbate.....	1: 1,000	3	4	3.01	94.3
			5	3.45	108.1
			8	4.05	126.9
			10	4.60	144.2
			13	4.78	149.8
			15	5.21	163.3
			18	5.27	165.2
			20	5.41	169.5
			23	5.54	173.6
			25	5.62	176.1
			28	5.18	162.3
			30	5.12	160.5
		Experiment 4			
		..	0	2.27	100.0
Histamine hydrochloride..	1:50,000	2	3	0.22	9.6
Epinephrine ascorbate.....	1: 1,000	4	5	1.28	56.3
			6	2.34	103.0
			8	2.05	90.3
			10	2.55	112.3
			13	2.78	122.4
Histamine hydrochloride..	1:50,000	14	15	3.13	137.8
			18	3.70	162.9
			20	4.15	182.8
			23	4.13	181.9
			25	5.20	229.0
			27	4.98	219.3
			30	3.30	233.4

sodium ascorbate. This is significant in view of the conflicting claims made for calcium in the treatment of asthma. It appears from this experiment that the synergistic effect of vitamin C on calcium may be the all-deciding factor in the therapeutic value of calcium in allergy. In fact, calcium gluconate may produce unfavorable results in the treatment of asthma, while calcium ascorbate may be useful.

GROUP 4.—Controls on this day showed fairly normal maintenance of the bronchiolar lumen, as well as a prompt response to a second dose of histamine hydrochloride after twenty-seven minutes.

Experiments 3 and 4: The histamine antagonism of benzedrine ascorbate was followed by active dilatation of the bronchiole. That this dilatation was active was demonstrated by a secondary response to

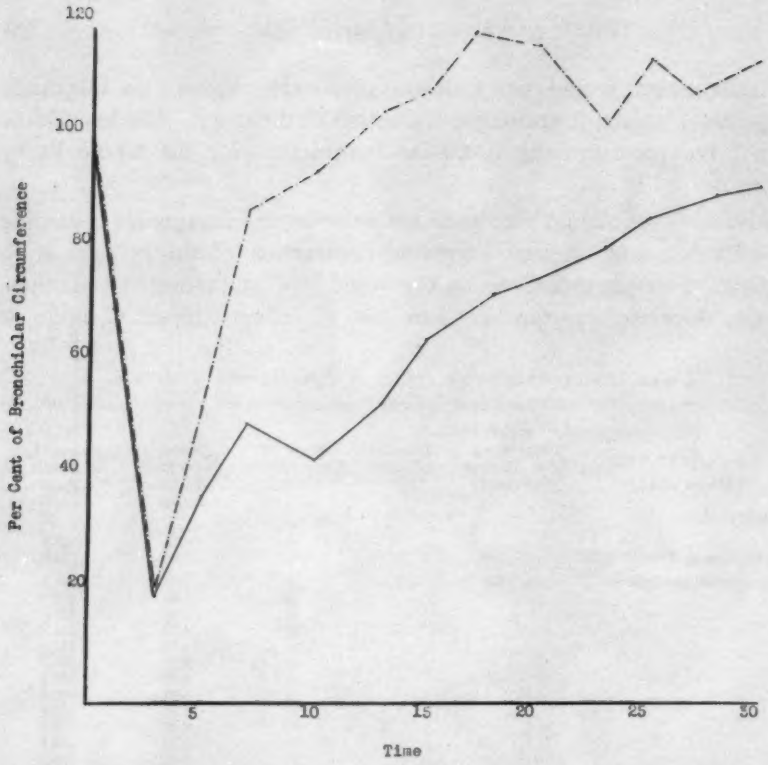

Chart 12 (group 5).—Histamine-antagonistic action of epinephrine hydrochloride. Experiment 3: Histamine hydrochloride, one minute; epinephrine hydrochloride, four minutes; histamine hydrochloride, fourteen minutes (solid line). Experiment 4: Histamine hydrochloride, one minute; epinephrine hydrochloride, four minutes (line of dots and dashes).

TABLE 12.—*Antihistamine Effect of Epinephrine Hydrochloride*

Test Substance	Final Concentration of Test Substance	Time of Addition (Min.)	Time of Observation (Min.)	Area, Arbitrary Units	Percentage of Original Area
Group 5		Experiment 5			
		..	0	3.47	100.0
Histamine hydrochloride..	1:50,000	1	3	0.67	19.3
Epinephrine hydrochloride	1:1,000	4	5	1.26	36.3
			7	1.73	49.8
			10	1.84	43.0
			13	2.38	53.0
Histamine hydrochloride..	1:50,000	14	15	2.23	64.2
			18	2.52	72.6
			20	2.62	75.5
			23	2.82	81.2
			25	3.00	86.4
			28	3.16	91.0
			30	3.21	92.5
		Experiment 6			
		..	0	4.43	100.0
Histamine hydrochloride..	1:50,000	1	3	0.91	20.5
Epinephrine hydrochloride	1:1,000	4	5	2.32	52.3
			7	3.89	87.8
			10	4.19	94.5
			13	4.68	105.6
			15	4.83	109.0
			17	5.05	119.9
			20	5.22	117.8
			23	4.60	103.8
			25	5.11	115.3
			27	4.84	109.2
			30	5.10	115.1

histamine, although the secondary contraction brought the lumen to only a little below normal, and well above the initial histamine contraction.

Experiments 5 and 6: These tests were all the more interesting because benzedrine sulfate gave no such response as did benzedrine ascorbate; in fact, it prolonged histamine contraction and produced virtually no histamine block. Benzedrine sulfate may be considered as bronchiole constricting.

Summary.—In this group of experiments a remarkable difference in pharmacologic action is evident. While benzedrine ascorbate produced a quick recovery from histamine contraction, with strong histamine block, the benzedrine sulfate showed no histamine antagonism, and in fact prolonged histamine contraction. The implications of this experiment may be important in relation to histamine shock. While benzedrine sulfate can keep a soldier alert, it may predispose him to greater histamine shock, whereas the vitamin C salt may protect against histamine shock.

GROUP 5.—Experiments 1 and 2: The bronchiole used in these controls was sensitive and reacted strongly to histamine, with poor spontaneous recovery; after twenty-one minutes the bronchiole responded with sharp contraction to histamine. The spontaneous recovery from histamine was only about 44 per cent.

Experiments 3 and 4: Epinephrine ascorbate produced immediate recovery from histamine, with such strong histamine antagonism that the second dose of histamine hydrochloride did not prevent continuation of the bronchiolar dilatation to over twice the normal size.

Experiments 5 and 6: Epinephrine hydrochloride, as was to be expected, produced active recovery from histamine contraction, with moderate dilatation of the bronchiole.

Summary.—One is struck by the remarkable synergistic effect of vitamin C on epinephrine. The epinephrine ascorbate showed about twice the bronchiole-dilating capacity exerted by epinephrine hydrochloride and a much quicker and more active histamine antagonism.

COMMENT

When all five groups of experiments are considered, it may be said that vitamin C (ascorbic acid) has slight histamine antagonism in itself, but when serving as the acid radical for calcium, ephedrine, benzedrine or epinephrine, it enhances their histamine antagonism and bronchiole-dilating capacity. This is essentially important in increasing respiratory ability.

Ephedrine sulfate and ephedrine hydrochloride constrict the bronchioles, while ephedrine ascorbate appears to have some dilating action.

Calcium gluconate exhibits no histamine antagonism; rather, it produces contraction of the bronchiole, while calcium ascorbate shows some antihistamine action, more than does vitamin C alone or sodium ascorbate. Benzedrine sulfate shows no histamine antagonism, but rather prolongation of the histamine effect, whereas benzedrine ascorbate displays pronounced histamine antagonism and bronchiole-dilating properties. Epinephrine hydrochloride shows, of course, strong histamine antagonism, but is only about one-half as active as epinephrine ascorbate, which actually dilated the bronchiole to almost twice that produced by epinephrine hydrochloride.

The observations on bronchiolar reactions by the technic here described likewise offer the opportunity for the study of ciliary action, as well as bronchial and bronchiolar rhythm. This, however, will require a study of longitudinal sections, rather than the cross sections here employed.

Sollmann and Gilbert [2] made the interesting observation that the movements of the bronchiolar cilia are not affected by most of the drugs that act on bronchiolar muscle. These authors stated that benzedrine is a constricting agent. This is to be attributed to the fact that they used benzedrine sulfate. The change in effect produced by the influence of the ascorbic radical is striking. They also found ephedrine to have but little effect. This is in agreement with the observation here recorded.

The observations in the experiments reported here seem to indicate that the acid radical associated with the ephedrine determines to a large extent the ephedrine action itself. Since these experiments were all conducted on rabbits, a basis of comparison of the action on the bronchioles of other animals must await further experimentation. Sollmann and Gilbert reviewed the work of Trendelenburg, in 1912; Jackson,[3] in 1913; Macht and Ting,[4] in 1921; Macklin,[5] in 1929; Swanson and Webster in 1930; Barlow and Beams,[6] in 1933; Cameron and Tainter,[7]

3. Jackson, D. E.: The Peripheral Action of Certain Drugs with Special Reference to the Lungs, J. Pharmacol. & Exper. Therap. **4**:291, 1913.

4. Macht, D. I., and Ting, G. C.: Response to Drugs of Excised Bronchi from Normal and Diseased Animals, J. Pharmacol. & Exper. Therap. **18**:111, 1921.

5. Macklin, C. C.: Musculature of Bronchi and Lungs, Physiol. Rev. **9**:1, 1929.

6. Barlow, O. W., and Beams, A. J.: A Comparison of the Bronchodilating Action of Several Anti-Asthmatic Agents After Anaphylaxis and Histamine Shock in the Guinea Pig, J. Pharmacol. & Exper. Therap. **47**:111, 1933.

7. Cameron, W. M., and Tainter, M. L.: Comparative Actions of Sympathomimetic Compounds: Broncho-Dilator Actions in Bronchial Spasm Induced by Histamine, J. Pharmacol. & Exper. Therap. **57**:152, 1936.

in 1936, and Co Tui, Burstein and Wright,[8] in 1936, found, however, that there was a rather close relationship between the results of the various investigators in regard to histamine response.

CONCLUSIONS AND OBSERVATIONS

It becomes apparent that vitamin C plays a unique physiologic role, which, in some respects, such as in the instance of epinephrine, is perhaps a sparing action on the body reserves through enhancement of activity. In the case of calcium the vitamin evidently plays an inherent role in the physiologic activities of calcium, as well as in its chemical relationship.

How important such a role could be is realized when the question of histamine shock is considered. In the presence of vitamin C deficiency the therapeutic use of calcium might even be contraindicated, whereas in the presence of adequate availability of the vitamin it could be an aid in the treatment of surgical shock. The use of the calcium ascorbate assures the desired physiologic response.

In view of the common use of calcium with adrenal cortex extract in management of surgical shock, this experimental demonstration of the difference between the action of the vitamin C salt of calcium and that of calcium gluconate becomes particularly important.

In my previous publications [9] I have suggested that calcium ascorbate serves as that part of the serum calcium compound in which the diffusible calcium occurs. The organic radical to which serum calcium is bound is likely to be the ascorbate rather than the citrate radical, hitherto proposed. The parallel action of calcium and vitamin C, which I have described, lends further support to this intimate relationship.

In the interest of brevity, the daily control charts and tables for groups 2, 3, 4 and 5 have been omitted.

Mrs. Madeleine L. FitzGerald and Mr. Saul Francis, technicians, made the preparations and the camera lucida drawings of the bronchioles, and Dr. H. D. Pease made available his laboratory facilities.

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8. Co Tui; Burstein, C. L., and Wright, A. M.: The Effect of Sympathectomy on the Sensitivity to Adrenalin of the Bronchioles, J. Pharmacol. & Exper. Therap. **58**:33, 1936.

9. Ruskin, S. L.: Studies on Parallel Action of Vitamin C and Calcium, Am. J. Digest. Dis. **5**:408, 1938; Studies in Calcium Metabolism: Further Contribution to Comparative Studies of Physico-Chemical Properties of Gluconate and Cevitamate of Calcium and of Vitamin C, ibid. **5**:676, 1938; Contribution to Study of Grippe Otitis, Myringitis Bullosa Hemorrhagica and Its Relationship to Latent Scurvy, Laryngoscope **48**:327, 1938; Calcium Cevitamate in the Treatment of Acute Rhinitis, Ann. Otol., Rhin. & Laryng. **47**:502, 1938. Ruskin, S. L., and Silberstein, M.: The Influence of Vitamin C on the Therapeutic Activity of Bismuth, Antimony and the Arsenic Group of Metals, M. Rec. **153**:327, 1941. Jonnard, R., and Ruskin, S. L.: Etude physico.- comparée du gluconate, du sel de calcium de la vitamine C et de la vitamine C, Compt. rend. Soc. de biol. **28**:266, 1938.

A SIMPLE METHOD OF MEASURING PERCENTAGE OF CAPACITY FOR HEARING SPEECH

FUNDAMENTAL FACTORS IN SETTING UP A STANDARD

EDMUND PRINCE FOWLER, M.D.
NEW YORK

Up to the present there has been no accepted standard for determining the percentage of loss of hearing. The Consultants on Audiometer and Hearing Aids of the Council on Physical Therapy of the American Medical Association have been criticized for not quickly producing a standard method of figuring percentage of hearing loss for use in industry. Some of the difficulties are apparent, and others are not generally recognized.

Every one with a hearing handicap wants to know its degree. "How much have I lost?" or "How much have I gained?" is the question. If the physician tells a patient his hearing is so many decibels down on average, or at certain frequencies, he is accused of speaking gibberish. It might as well be Sanskrit for all the information it imparts. What every one wants to know is the "percentage of hearing" lost. The patient can understand percentages, and thinks he can understand percentage of hearing loss. At any rate, percentage figures should give him a clear answer to the question, "Has the treatment really improved my hearing?"

At first thought it might appear that the simplest standard would be to use a definite volume of speech and to measure the distance it is heard. But the problem is not so simple as this, because it is difficult to speak at a given intensity using natural speech,[1] and artificial speech, such as that from a phonograph, is not usable except as a rough gage.

The main difficulty in setting up a standard for measuring the capacity to hear speech lies in the fact that speech is made up of many varying combinations of frequencies, consisting of broad and narrow frequency bands, all of which differ in intensity and importance under

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Read before the Section on Laryngology, Otology and Rhinology at the Ninety-Third Annual Session of the American Medical Association, Atlantic City, N. J., June 12, 1942.

1. Fowler, E. P., Jr.: Extraneous Factors in Quantitative Tests for Hearing, Acta oto-laryng. **28**:283-304, 1940.

different conditions of production and reception. If a man is accustomed to hearing conversational speech, with a great or total loss of hearing for some frequencies, all the frequencies he does hear assume a different importance than they do for a normal person.

Most of the vowel sounds are relatively low in frequency and high in intensity. Most of the consonants are relatively high in frequency, and both vowels and consonants vary in intensity and frequency with different people. Some speech sounds are explosive in character. The consonants and sibilants do not have a definite frequency; they consist of breath, hissing or buzzing sounds. In other words, they consist of bands of frequencies. This last is in a way fortunate because in spite of loss of hearing for a portion of the speech frequencies, one can often sense their frequency content through acute perception of the other frequency patterns which are normally associated with them. This mental interpolation is a physiologic phenomenon observed in normal people when they listen to very faint speech or to any speech at a great distance. There are great individual variations in all these phenomena. Loud speech accentuates the low frequencies more than the high frequencies. One can often miss hearing many articulations, and even several words, in a sentence without missing the meaning. These phenomena can be compensated for by weighting the frequencies according to their average relative usefulness in hearing speech. Measuring the over-all decibel intensity of speech does not make allowances for any of these phenomena, because it measures the loudness and not the clarity. It is therefore unreliable for ascertaining the capacity to hear speech.

When the lower frequencies (up to about 300) are experimentally filtered out, there is a loss of only about 2 per cent of articulation when unrelated words are used. When related words are used, as in conversation, the hearing loss is only a fraction of 1 per cent. This means that the frequencies of 256 (middle C on the piano) and below are relatively of little importance in hearing speech. When the higher frequencies (above 4000) are filtered out, or when for any reason a person cannot hear the frequencies over 4000, there is a loss of less than 1 per cent in the hearing for words. For these reasons it is necessary to use only four frequencies between these relatively unimportant high and low areas. These four frequency areas (512, 1024, 2048 and 4096) are the most important for hearing speech, but they differ in importance, and therefore I have assigned to them a weighting figure derived both from the experimental and from my clinical observations. An exact percentage of importance has been determined by Fletcher and Steinberg and others by filtering out each frequency region in turn and measuring the resultant loss in articulation. Also,

I have determined the percentage of importance by testing patients whose audiograms showed marked narrow "troughs" at these frequency regions. The two methods check remarkably well. My choice was influenced somewhat by expediency, for uneven numbers in the teens are awkward for mental multiplication and the difference in any case is not more than 1 or 2 per cent. The numbers 15, 30, 40 and 15 are all easily used for multiplying the decibel loss at each of the four frequencies respectively, no matter what that loss proves to be.

People normally use both ears to hear speech, but if one ear hears better than its mate, the poorer ear is used more for sensing the mere presence of speech than for the articulation. However, the poorer ear is still useful, for it aids in the localization of speech and in picking up warning sounds coming from its side of the head. My tests show that two ears hear better than one, even when one is much deafer than the other. The percentage of loss of hearing for speech should therefore always be based on the binaural hearing capacity for speech at levels well over threshold. In small rooms ordinary speech often reaches the better ear in sufficient intensity for understanding even if it is turned away from the speaker, as a result of reflections from adjacent walls or by conduction through or around the head. In large rooms these reflection sounds, particularly the consonants, are often too weak for intelligibility.

It is well known that two persons with similar threshold audiograms frequently do not hear speech alike. The chief reason for this discrepancy is that in nerve deafness the loss is not linear, owing to the ever present recruitment phenomenon, which enables persons with this type of deafness to hear sounds well over threshold nearly or just as loud as a normal person, whereas persons with obstructive deafness never hear a sound as loud as does a normal one. In obstructive deafness the loss is linear.

However, recruitment in severe nerve deafness is accompanied by loss in clarity, so that for losses of over 40 decibels there is a progressive loss in clarity. This is the "blurring phenomenon" so prominent in severe nerve deafness.

To complicate the picture still further, when hearing for the speech frequencies is down over 45 or 50 decibels because of neural lesions, another phenomenon turns up from time to time which must be taken care of in evaluating the capacity to hear speech. This is the phenomenon of marked or total deafness for speech without a corresponding loss of hearing for the speech frequencies and without any loss of memory for speech. The speech frequencies are heard fairly well, but their patterns cannot be recognized with sufficient clarity to be understandable.

Another difficulty in setting up standards for the measurement of sensations, such as hearing, sight, smell, balance, taste and touch, is that sensations do not necessarily correspond to the degree of stimulation which provokes them. However, one can set up average figures, which are far better than no standard at all. This I have done, with the understanding that the standard is to be tentative. [2]

METHOD

All of these factors should be taken into consideration in preparing a standard for measuring the percentage of capacity for hearing speech. The method I propose does this. It is carried out as follows (table 1):

1. Test each ear with an accepted standard audiometer and plot the air and bone conduction losses in the approved manner. (Use a soundproof room.)

2. Multiply the air conduction loss at the following frequencies by the corresponding weighting factors:

Frequencies	512	1024	2046	4098
Weighting factors, %	15	30	40	15

3. Add the products and divide by 100 (by moving the decimal point two figures to the left). This is the weighted decibel loss when there is no nerve deafness.

4. If there is a bone conduction loss of 15 decibels or more, apply the designated recruitment factor before averaging the air conduction loss.

To use the Fowler table (table 1) for estimating the percentage loss, proceed as follows:

1. Drop a vertical line from the figure representing the weighted decibel loss in the better ear.

2. Draw a horizontal line from the figure representing the weighted decibel loss in the worse ear. The point where these vertical and horizontal lines intersect indicates the percentage loss.

It will be observed that:

1. Each ear is tested individually, and the binaural hearing capacity is obtained by a changing ratio according to the degree and difference in the losses in the better and in the worse ear.

2. An accepted audiometer is used to obtain the threshold air and bone conduction losses at each frequency, and a standard audiogram chart is used for plotting.

3. The hearing capacity is considered dependent on what a person hears binaurally by air-borne sound without artificial aids.

2. All these matters have been discussed in detail in previous papers.

4. The frequencies are weighted according to their importance for speech.[3]

5. Changing increments of loss in hearing capacity are assigned to slight, moderate and severe deafness.

6. Allowances are made for unequal binaural losses and for monaural total deafness.

7. A weighted loss of 100 decibels is considered a total loss of hearing for speech.

TABLE 1.—*Fowler's Table for Estimating Percentage Loss of Capacity for Hearing Speech in Monaural and Binaural Deafness*

Weighted Decibel Loss—Worse Ear	Weighted Decibel Loss—Better Ear										
	0	10	20	30	40	50	60	70	80	90	100
0	0%	0	1	5	9	14	20	27	35	44	54
10	0	2	3	6	10	15	21	28	36	45	55
20	1	3	4	7	11	16	22	29	37	46	56
30	2	4	12	20	27	35	44	54	63	71	78
40	3	5	14	24	35	44	54	63	71	78	84
50	4	6	15	27	40	54	63	71	78	84	89
60	5	7	16	29	44	59	71	78	84	89	93
70	6	8	17	30	47	63	76	84	89	93	96
80	7	9	18	31	49	66	80	88	93	96	98
90	8	10	19	32	50	68	83	91	96	98	99
100	9%	11	20	33	51	69	85	93	97	99	100%

Percentage Loss of Capacity

Recruitment Factor

Apply corrections before averaging A.C. losses

Subtract from A.C. loss, for B.C. loss <15 db., 0
15 5
20 4
25 3
30 2
35 1
40 0

Add to A.C. loss, for B. C. loss = 45 2
50 5
Over 50 5

Weighted decibel loss is the sum of the corrected A.C. losses at the following frequencies, times the corresponding percentage factors:

512	1,024	2,048	4,096
15%	30%	40%	15%

The percentage loss will be indicated at the intersection of a vertical line from decibel loss in better ear with a horizontal line from decibel loss in worse ear

(Test in a soundproof room)

A.C. indicates air conduction, and B.C., bone conduction.

It will also be observed that the losses at four octave intervals are used to represent the average loss for the frequencies one-half octave over and one-half octave below them. This is simple and renders it possible to use all the frequencies from about 400 to 6,000—in other words, to include all the frequencies necessary for perfect hearing for speech and yet to use only four frequencies in the calculations, that is, 512, 1024, 2048 and 4096.

I have set forth at length all these factors because in adopting any standard I believe it is essential to understand the difficulties and limitations encountered. I have given much thought to this subject

3. I have discussed in previous papers the Fletcher, Steinberg and Fowler weightings and the reasons for using them. Except for neural types of hearing loss these weightings work out much the same.

for many years and have endeavored to encompass in a simple method all the factors involved. Some of the important factors are now generally acknowledged, but the question is which should be included in a standard acceptable to all concerned. Some of the factors are not generally understood, and for this reason it may be necessary to defer their inclusion until a later date.

I must stress the point that my standard is an over-all standard. If some one wishes to set up a method for measuring the capacity to hear speech at some near distance, say within 5 feet (150 cm.), then my table should be foreshortened and the figures of percentage loss lowered. However, if this is done it must be understood that the standard is a very limited one. Without special effort people do not limit their speaking distance to 5 or 4 feet (150 or 120 cm.) or to any other short distance.

The following factors appear to be most puzzling: (1) the basis for assigning the weightings to each of the speech frequencies; (2) the basis for taking care of all combinations of nerve deafness and obstructive deafness, i. e., the basis for using the recruitment factor; (3) the changing binaural ratio of loss with slight and pronounced differences in hearing in the two ears; (4) the decibel loss which best represents a total loss of hearing, and (5) speech deafness out of proportion to the loss of hearing for the speech frequencies. These factors are all real, and therefore they should be admitted and discussed.

COMMENT

With the exception of the more even gradients and the heavier weighting for severe degrees of deafness (40 to 70 weighted decibel loss), the percentages of hearing loss determined by several other schemes will not differ from mine by more than 10 to 15 per cent when the deafness is due to obstructive lesions. However, in cases of nerve deafness there will be differences up to 20 per cent or more. In explaining the causes for these great differences, I shall discuss the five factors which seem to be less generally understood.

My table was adopted after many years of clinical observation and after discarding many other schemes, all of which failed to obtain percentage values which corresponded closely with clinical experience. The hearing of all of my patients was computed and compared in this way. However, other facts substantiate my figures. For instance, table 2 shows the estimates of 361 persons as to the relative importance that should be given to near conversation, telephone speech and far speech, such as that heard at public addresses (suggested in part by Mr. Steinberg). Each person was also asked his opinion as to his own percentage of loss of hearing for speech.

Table 2 shows the distribution of various estimates made by the two groups of subjects (the hard of hearing and persons with normal hearing). Included in each interval are all percentage values from 0 to 9, 10 to 19, etc. For example, in the first column 2 hard of hearing persons estimated the value of near conversation to be 100 per cent; 3, from 90 to 99 per cent, and 23, from 80 to 89 per cent. No person estimated near conversation to be of as little value as 0 to 9 per cent.

In the summary in the lower part of the table, Q_1 means that one fourth of the subjects gave lower estimates than the midde of the distribution; M indicates about the middle (i. e., medium estimate) of

TABLE 2.—*Estimates of Three Hundred and Sixty-One Persons as to the Relative Importance of Three Categories of Speech* *

Estimated Percentage of Importance	Near Conversation		Telephone Speech		Far Speech (Over 15 Ft.; Public Addresses, Etc.)	
	Hard of Hearing	Normal	Hard of Hearing	Normal	Hard of Hearing	Normal
100	2	..	..	..	..	..
90 to 99	3	1	..	1	..	..
80 to 89	23	22	..	..	..	..
70 to 79	49	58	1	..	1	..
60 to 69	41	34	1	2	1	..
50 to 59	54	30	9	4	5	2
40 to 49	9	7	8	6	10	6
30 to 39	7	7	33	30	17	19
20 to 29	7	4	62	42	62	37
10 to 19	2	1	59	56	70	74
0 to 9	..	..	24	23	31	26
Total no. of persons	197	164	197	164	197	164
Q_1, %	50-59	50-59	10-20	10-20	10-20	10-20
M, %	60-69	60-69	20-30	20-30	10-20	10-20
Q_3, %	70-79	70-79	30-40	30-40	20-30	20-30
Range, %	10-100	10-90	0-70	0-90	0-70	0-50

* Prof. R. Pintner made the calculations in table 2.

the distribution, and Q_3, about the three-fourths point. The range is from the lowest to the highest estimate. In other words, for the first column ("Near Conversation") the medium estimate for the hard of hearing as to the relative importance of near speech fell between 60 and 69 per cent. This was also true of the persons with normal hearing. An interesting thing about the distribution for all three categories of speech is that both the normal and the hard of hearing made exactly the same kind of estimates; namely, the relative importance of near conversation was estimated to be about 65 per cent, as compared with 20 per cent for telephone speech and 15 per cent for far speech.

It is apparent from these data that if a person on the average cannot hear ordinary conversational speech at more than 4 to 5 feet (a distance usually possible in small rooms), there is an over-all loss of speech of only about 15 per cent if there is still good hearing for

telephone speech. Careful observations reveal that unless special effort is made, one third of the time a person will be more than 4 or 5 feet away from the speaker, with the net result that a person who can hear only at a distance of 5 feet will on the average really be hearing only two thirds of even the small room speech to which he is normally exposed. For this reason about one third of 65 per cent, say 20 per cent, may be deducted for this factor. If this is true, then even though telephone speech is well heard, there is a 35 per cent loss for speech, i. e., the 15 per cent assigned to all far speech plus the 20 per cent deducted from the 65 per cent assigned to all near speech.

Persons with a 40 decibel weighted loss cannot hear speech frequencies 30 decibels over threshold at 5 feet unless the intensity of the average conversational voice (45 decibels) is voluntarily or involuntarily raised in volume or the distance is reduced below 5 feet. In other words, it appears to follow that a 40 decibel weighted loss would correspond to a loss of about 35 per cent in capacity to hear speech. This is the exact figure assigned to such a loss in my table.

I chose a 40 decibel loss as an illustration because 40 decibel losses in obstructive and in nerve deafness are closely comparable, whereas in nerve deafness for a weighted loss of 45 or 50 decibels or more the blurring phenomenon enters the picture and for losses of less than 40 decibels the recruitment phenomenon lessens the loss indicated by the air conduction audiogram alone. All of these factors are taken care of in my method.

For many years I have asked hard of hearing people what they themselves estimated their loss of hearing for speech to be. The great majority of persons with an average weighted loss of 50 to 60 decibels in both ears thought they suffered a loss of from 55 to 70 per cent for speech. It will be noted that this distribution corresponds within 1 per cent to the percentage losses between the 50 and the 60 decibel weighted loss in my table. I assign to a binaural 50 decibel weighted loss a percentage loss of 54 and to a 60 decibel weighted loss a percentage loss of 71. These and the other figures in the table for estimating the percentage capacity for hearing speech were filled in to correspond with the weighted decibel losses as checked by clinical observation in hundreds of test cases. Unless there is close agreement with clinical observation no standard is dependable.

A 100 decibel weighted loss was chosen to represent a 100 per cent loss in capacity to hear speech because it does in fact do this; moreover, 100 decibels is a logical and convenient upper limit. All the approved audiometers include 100 decibel intensities at the four frequencies I have chosen. It will be noted that had I chosen 90 or 110 decibels as the upper limit the difference in percentage calculations would have suffered a 2 per cent shift at most.

Of more value than the guesses as to the relative importance of distant, near and telephone speech is the time people spend on the average in listening to each type of speech. I have discussed my data along these lines in previous papers. It is evident that age, occupation, social status, place of residence and many other factors are involved. My data show that on the average most people are in a position to hear fairly near speech seven to eight hours a day, far speech one to two hours a day and telephone speech one-half to one hour a day and that for four to five hours of the waking day they do not listen to speech. All these data have been used in determining my percentages of hearing loss.

How loud must speech be to be understood? According to Steinberg and Gardner's unrelated syllable articulation tests, if ordinary speech reaches the ear at 30 decibels over threshold, about 70 per cent of the speech sounds are heard and recognized. Intensities less than 30 decibels over threshold cause severe losses. (At 20 decibel intensity only 40 per cent of the articulations are recognized.)

To hear 70 per cent of the articulation is usually sufficient for ordinary conversation in home and business, for one does not have to hear every word in a sentence to understand what is said.

Figure 1 illustrates graphically the difference in loudness gained by a given intensity of the speech frequencies in obstructive and in nerve deafness and shows the limitations imposed by the intensities available for speech.

Since, as has already been shown, speech must be 30 decibels over threshold to be fairly understandable (i. e., to hear 70 per cent of the articulation), when there is obstructive deafness with a 40 decibel loss, speech frequencies on the average must reach the ear with an intensity of at least 70 decibels to be fairly understandable. For a threshold loss of 50 decibels they must reach the ear with an intensity of 80 decibels—in other words, the equivalent of a shouting voice near the listener. People do not like to shout, and so to make themselves heard they prefer to move to within 3 or 4 feet (90 or 120 cm.) of the deafened person. If there is a 70 to 80 decibel loss, no ordinary conversation will be comfortably heard or understood unless the speaker raises his voice or is less than 1 foot (30 cm.) away. There is then a 100 per cent loss for unamplified speech at distances greater than 1 foot.

In cases of nerve deafness, as shown by the diagram (fig. 1), much less intensity is required to reach a 30 decibel level above threshold. For instance, with a 40 decibel loss speech frequencies need reach the ear with an intensity of only 54 decibels (14 decibels over threshold) to make them seem as loud as an intensity of 70 decibels would to an ear with 40 decibels of obstructive deafness. There is a gain of 16

decibels in favor of the nerve-deafened ear, but even this is so near the threshold that the average 45 decibel intensity of ordinary speech will be heard only faintly (14 decibel loudness) at a distance of 4 or 5 feet. It should be borne in mind that just because a person hears what is said at 20 feet (610 cm.), or at any other distance, does not necessarily mean he is hearing speech at average normal intensity or that he is hearing 100 per cent of the articulation. If he has any hearing loss he is hearing speech at fainter than normal intensity. However, the recruitment phenomenon explains how one person may hear better than another even though the air conduction audiograms are similar.

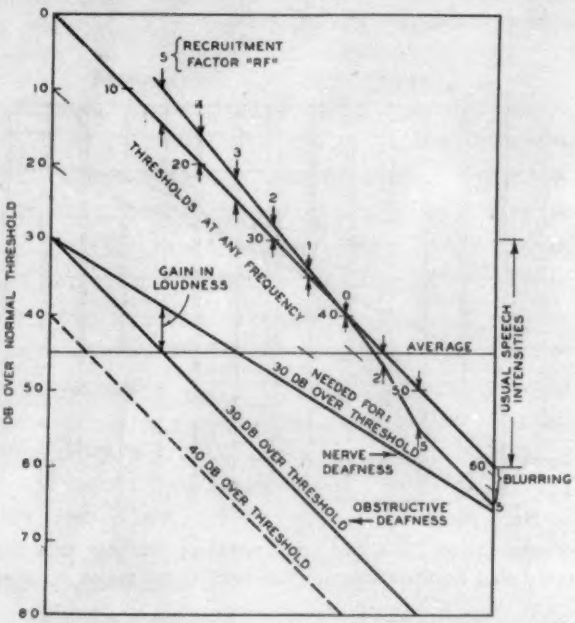

Fig. 1.—Relation of gain in loudness and speech intensity in nerve and obstructive deafness and effect of the recruitment factor.

These simple illustrations should suffice to prove the necessity of allowing for the recruitment factor in any standard for estimating the capacity to hear speech. One should remember that threshold sounds are not of much use for hearing speech and that in the presence of nerve deafness all sounds well above threshold are subject to the recruitment phenomenon.

With the lesser degrees of nerve deafness the recruitment phenomenon practically removes the handicap (fig. 1), but with severe deafness there is loss in clarity, so that for losses of over 40 decibels at any frequency the gain from the loudness is largely annulled by the loss in

clarity. Moreover, losses of over 40 decibels raise the threshold to such an extent that only loud speech can be clearly heard without artificial aid or near proximity to the sound source. It must always be kept in mind that it is not only the air conduction threshold value but the degree of nerve deafness and environmental noise which determines the efficacy of the gain in loudness from the recruitment phenomenon. It is not generally realized that in the presence of noise the normal, as well as the partially deafened, ear responds to speech sounds by "recruitment" because the noise temporarily causes a "busy wire" nerve deafness. Nerve deafness is best measured by charting bone conduction loss and the recruitment of loudness. These procedures also are of use in prescribing hearing aids.

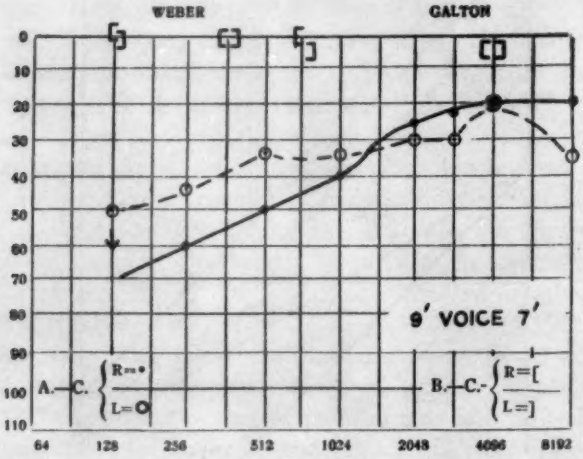

Fig. 2.—Computations in a case of obstructive deafness in a patient aged 18. Here it is noted that no allowance for the recruitment factor is necessary.

Right Ear A. C. Loss		Weighting		Weighted Db. Loss		Left Ear A. C. Loss		Weighting		Weighted Db. Loss
50	×	15	=	750	512	35	×	15	=	525
40	×	30	=	1200	1024	35	×	30	=	1050
25	×	40	=	1000	2048	30	×	40	=	1200
20	×	15	=	300	4096	20	×	15	=	300
				32.50						30.75

Percentage loss of capacity to hear speech 22.

In this figure, and in figures 3, 4 and 5, *A. C.* indicates air conduction.

Of course, as I have pointed out, even if certain frequencies are not heard loud enough for clear intelligibility in speech, the mere fact that they are heard at all is of some use in hearing speech; moreover,

they may serve to direct the attention to the source from which the speech sounds are emanating (stereophonic effect).

The allowance for unequal binaural losses and for monaural total deafness should be not a fixed ratio but a changing increment of percentage loss, according to increasing and decreasing differences in decibel loss between the better and the worse ear, since this is the way the two ears function binaurally. My table allows for such differences.

As I see it, a total loss of hearing for speech cannot be inferred from any loss at a single frequency because the percentage loss depends on the weighted decibel loss at all of the most important speech frequencies—in other words, on the total picture and not on one, two

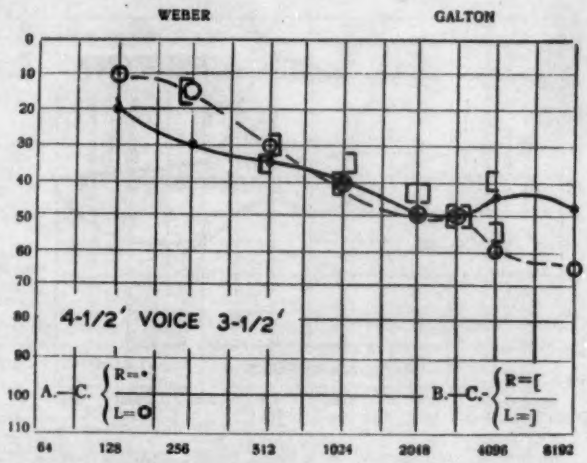

Fig. 3.—Computations in a case of nerve deafness in a person aged 65, showing allowance for the recruitment factor (R. F.).

Right Ear A. C. Loss	R. F.		Weighting		Weighted Db. Loss
35	— 1	= 34	× 15	=	510
40	— 0	= 40	× 30	=	1200
50	+ 2	= 52	× 40	=	2080
45	— 0	= 45	× 15	=	675
					44.65

Left Ear A. C. Loss	R. F.		Weighting		Weighted Db. Loss
30	— 2	= 28	× 15	=	420
40	— 1	= 39	× 30	=	1190
50	+ 2	= 52	× 40	=	2080
60	+ 5	= 65	× 15	=	975
					46.65

Percentage loss of capacity to hear speech 47. The soft voice was heard at about 3½ and 4½ feet (105 and 135 cm.) in the right and in the left ear, respectively.

or three narrow views of it. Therefore I include in my computations all losses up to 100 decibels. Over this it is not necessary to go because

it is out of range of the ordinary voice. It is often out of range of even the amplified voice.

I now call attention to a phenomenon which, as far as I know, has not heretofore been considered in estimating percentage loss for speech. I refer to a severe or total loss of intelligibility for speech without cortical lesions or sufficient losses at the speech frequencies to account for it. This factor cannot be ignored.[4] The air conduction audiogram is useless to detect it. The bone conduction audiogram is a valuable guide to the possibility of its presence, and in part to its degree. However, I believe actual speech should be used to detect or eliminate this type of severe deafness, and when it is found the ear so affected should be considered as a totally deaf ear and treated accordingly in

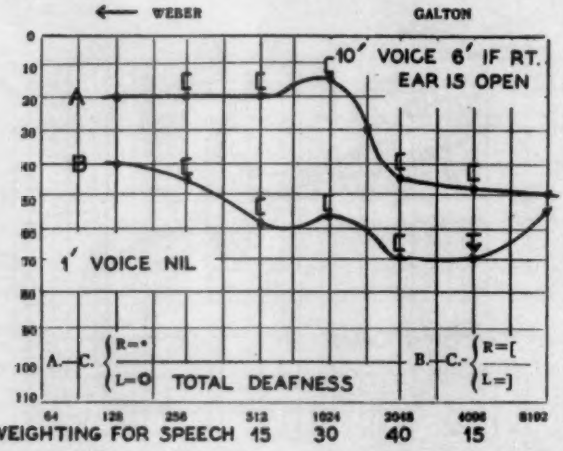

Fig. 4.—Computations in the case of a child whose hearing varied from time to time, as a result of nerve lesions, and whose left ear was found to be totally deaf at all examinations. Hearing in the right ear is shown at its best (*A*) and at its worst (*B*).

A Right Ear A. C. Loss	R. F.	Weighting	Weighted Db. Loss		*B* Left Ear A. C. Loss	R. F.	Weighting	Weighted Db. Loss
20 — 5 =	512	× 15 =	225		60 + 5 =	512	× 15 =	975
15 — 0 =	1024	× 30 =	450		55 + 5 =	1024	× 30 =	1800
45 — 0 =	2048	× 40 =	1800		70 + 5 =	2048	× 40 =	3000
45 + 2 =	4096	× 15 =	705		70 + 5 =	4096	× 15 =	1125
			31.80					69.00

Percentage loss of capacity to hear speech *A* 37, *B* 92. The child heard at 10 feet (3 meters).

4. Reported on at the Seventy-Fifth Annual Meeting of the American Otological Society, Atlantic City, N. J., May 28, 1942.

calculating percentage loss for speech. It, certainly, is of little or no use for hearing speech no matter how well it hears the speech frequencies.

In studying any method for determining the percentage of loss in capacity to hear speech, it is important to keep in mind that one unconsciously speaks louder to persons one knows to be deafened, and in noisy surroundings, and that many people are so alert and competent, and such good lip readers, that they appear to hear much better than is really the case. Moreover, one's estimation of the hearing ability

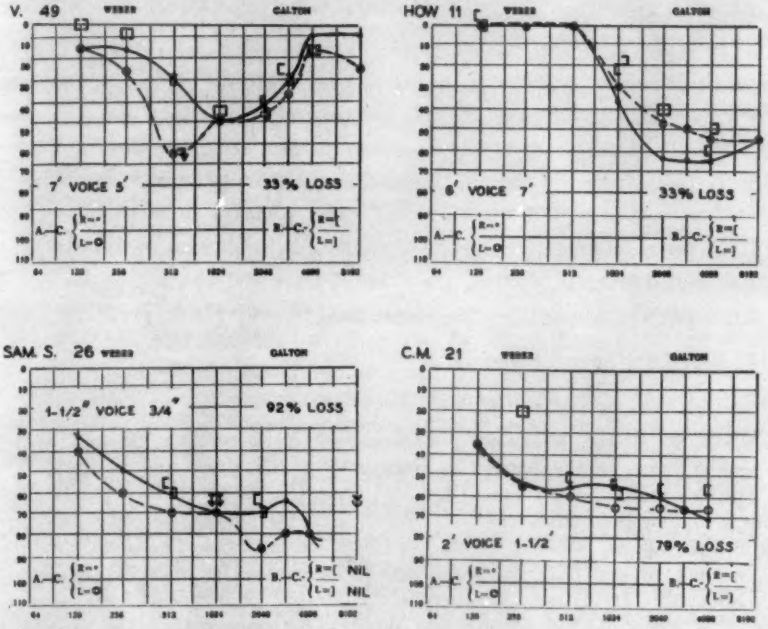

Fig. 5.—Audiograms in 4 cases of almost pure nerve deafness. The distances at which the soft conversational voice was heard and understood show that the percentage losses of capacity as obtained with my table in each instance were really not excessive, although greatly more than those allowed by methods not using my standards, often as much as 20 per cent more.

of a hard of hearing person is often exaggerated because one does not appreciate the increase in hearing brought about by his hearing aid or because one does not know that an aid is being used. The percentages obtained by the use of my standard test, therefore, will often at first sight appear too high (figs. 2, 3, 4 and 5).

My method is designed for an over-all estimate, i. e., for far speech, near speech and speech heard at all intermediate distances, not just

for speech heard at a distance of 4 or 5 feet.[5] I have not discussed the psychologic and mental variations encountered in different types of hearing loss because these should be judged on their merits, by courts or other proper authority.

CONCLUSIONS

Constant clinical use has proved that the standard I have set up is reasonable and dependable under the conditions described and, most important, that it takes account of the following factors:

1. It makes a more sensible and simple allowance than other methods for the relative importance of the speech frequencies in understanding speech.

2. It takes equitable care of the changing binaural ratio of hearing loss with different degrees of deafness and with slight and pronounced differences in the hearing of the two ears. No other method does this.

3. It takes care of the discrepancies in hearing speech in cases of nerve and conduction deafness. No other method does this.

4. It takes cognizance of the considered judgment of hundreds of normal and hard of hearing persons.

Any method which ignores these factors will cause great injustice.

140 East Fifty-Fourth Street.

DISCUSSION

Dr. W. E. Grove, Milwaukee: Measurement of a person's capacity to hear the spoken voice by any method is fraught with difficulties, and in my opinion Dr. Fowler's method is not simple, although it may be as simple as it can be made. There is no simple method. As. Dr. Fowler says, the average person is interested only in how well he can hear the spoken voice. In most cases, he is not interested in how well he can hear by bone conduction because he does his hearing by air conduction. He is not interested in the niceties of diagnosis. He wants to know how much hearing he has lost through the only medium of hearing he understands, namely, air conduction. The ideal test for determining this loss would be one utilizing the human voice; the limitations of such a method are well known. The next most feasible method would be one utilizing a reproduction of the human voice on a phonograph record, but this too presents its difficulties, such as the accompanying noise of the operation, the rapid deterioration of the records and the number of recordings necessary to produce all types of the human voice.

The accepted pure tone audiometer is the nearest approach to an instrument of precision available for hearing tests with the exception of properly calibrated tuning forks. To be sure, the audiometer does not test the capacity to hear speech, but it does indicate the subject's capacity to hear pure tones over the range covered

5. I hazard the opinion that for ordinary conversational speech up to 5 feet (150 cm.) should be considered near, 5 to 15 feet (150 to 450 cm.) middle hearing distance and over 15 feet far hearing distance.

by speech, roughly from 300 to 4000 cycles. Dr. Fowler states that different persons with the same audiometric curve for air conduction hear differently. This may be true; I have no way of verifying that suspicion, because the only other test agent with which I could verify it, my own voice, is not sufficiently accurate to check on the audiometer.

Now, as Dr. Fowler has pointed out, all portions of the audiometric field covering the range of the voice, the speech frequencies from 256 to 4000 cycles and from threshold to about an 80 or 90 decibel loss, are not of equal importance for understanding of speech. For this reason the decibel readings on the audiometric chart must be weighted in accordance with the relative importance of various portions of the field. In other words, the tones 512, 1024 and 2048 double vibrations are much more important in the capacity to hear speech than those below 512 or above 2048 double vibrations, and consequently the losses exhibited for these three tones must be given higher percentage values than are given to other tones. That is what is meant by decibel weighting.

There are other difficulties inherent in setting up a percentage table for loss of hearing. A table set up to cover all classes of persons and all vocations of life would not and could not operate equally for all. For instance, a hearing loss which might not impair the efficiency of a laborer and which might even be advantageous to a worker in noisy surroundings would constitute a much higher disability for a musician, a telephone operator, a piano tuner, a stenographer or a proofreader. A single basic table cannot be made to fit all persons and all occupations, just as a visual loss according to the table now in use does not represent the same loss of capacity to see in all cases. However, as Dr. Fowler has put it, "one can set up average figures which will be far better than no standard at all."

DR. DOUGLAS MACFARLAN, Philadelphia: I want to preface my remarks by saying that the opinion I express is not the opinion of the committee interested in this matter of hearing, the subcommittee of the Council on Physical Therapy of the American Medical Association.

I have been giving tests of speech hearing to practically every patient for twenty-five years, using a phonograph audiometer with a decibel meter and a single intensity record. What Dr. Fowler says about the variability of intensities and frequencies in speech sounds is true. Words differ considerably, not only in intensities and frequencies but in interpretability. Using single or paired numbers, one can get a remarkably efficient test of the hearing of words. The variability between the hearing of these numbers usually does not amount to 10 decibels, and this is no more than the "observational error."

Until Dr. Fowler insisted on the weighting of frequencies it was impossible to translate the results of tests for frequency hearing into terms of true efficiency of speech hearing. The method first advocated by Fletcher was grossly erroneous. Now, with the weighting of discrete frequencies, this translation is possible with fair accuracy and the records with the oscillator and those with the phonograph compare fairly well. My experience with the phonograph audiometer tells me more about speech hearing than frequency audiometry ever does. I feel that it is more accurate for estimating speech hearing. This conclusion has been reached after years of simultaneous use of the two methods. Speech is so much more subtle and more complicated than are single tones that it can be tested with real efficiency only by the use of speech itself as the test stimulus.

My plea is that otologists use the phonograph routinely. I am sure that they will come to believe as I do.

DR. EDMUND P. FOWLER, New York: In regard to Dr. Grove's discussion, it is true that persons do not normally hear much with bone conduction, but when they have much obstructive deafness they hear by both air and bone conduction.

As to the importance of the frequencies, this varies, depending on whether or not a person has normal hearing. If a person hears perfectly all the frequencies from 128 up to, say, 16,000, then of course the usual weighting allowances are very good. But for a person who is born with a "dip" at the top of the scale, who never did hear the high tones, the relative weighting is quite different, and so a compromise in the weighting of figures is necessary in order for these figures to cover all or almost all conditions.

In regard to Dr. Macfarlan's remarks as to the recording of speech intensity, I repeat, intensity alone is very misleading. One often hears a person say: "Oh, I hear your voice, but I can't hear what you say." Such a person is getting sufficient intensity but no speech.

STAPES, FISSULA ANTE FENESTRAM AND ASSOCIATED STRUCTURES IN MAN

III. FROM EMBRYOS 6.7 TO 50 MM. IN LENGTH

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In earlier articles[1] the major steps in the rapid embryologic development of the stapes and those in the slower alteration of postnatal specimens were described and illustrated, selected stages between 7½ weeks (22.8 mm.) and 70 years being utilized; the morphologic aspects of the closely related vestibular rim and fissular prolongation were considered at the same time. In the present phase of the investigation a more inclusive and numerically larger set of stages is being studied, ranging between that of the embryo 6.7 mm. in length and that of an adult aged 75 years. By this means, detailed information on the morphogenesis and increment will be added, and hiatuses in present knowledge of stapedial form will be closed.

The current article will carry the stapedial history through stages at which the cartilage has assumed definitely stirrup-shaped form; subsequent articles will account separately for equally crucial morphogenetic epochs.

MATERIAL AND METHODS

Twenty-six embryonal series in the Carnegie collection were examined, and photomicrographs of representative levels were obtained in twelve specimens. Four fetal series in the Wisconsin collection were studied and photographed. All

From the Department of Anatomy and the Department of Otolaryngology of Northwestern University Medical School. Contribution no. 372 from the Anatomical Laboratory.

Dr. George H. Corner, of the Carnegie Laboratory, Baltimore, and Dr. T. H. Bast, of the department of anatomy of the University of Wisconsin, permitted us to study their excellent series.

Presented as part of an exhibit at the 1941 meeting of the Academy of Ophthalmology and Otolaryngology, in Chicago. Read at the 1942 meeting of the American Association of Anatomists, in New York. A research conducted under the auspices of the Central Bureau of Research of the American Otological Society.

1. Anson, B. J.; Karabin, J. E., and Martin, J.: (*a*) Stapes, Fissula Ante Fenestram and Associated Structures in Man: I. From Embryo of Seven Weeks to That of Twenty-One Weeks, Arch. Otolaryng. **28**:676-697 (Nov.) 1938; (*b*) II. From Fetus at Term to Adult of Seventy, ibid. **29**:939-973 (June) 1939.

photographs bearing the Carnegie catalogue number were obtained through the generosity of Dr. Corner. Sections from the following specimens are illustrated:

Length (Crown-Rump)	Catalogue No.	Series		Figure	Original Magnification	Thickness, Microns
9.6 mm.	Carnegie 6517	12.2.4	(L)	1	85	8
9.6 mm.	Carnegie 6517	12.3.1	(L)	2	85	8
10.6 mm.	Carnegie 6524	20.1.1	(R)	3	85	10
10.6 mm.	Carnegie 6524	20.3.2	(R)	4	85	10
12.9 mm.	Carnegie 6520	20.1.5	(R)	5	85	10
13.1 mm.	Carnegie 6527	18.2.3	(R)	6	85	15
13.95 mm.	Carnegie 4430	11.2.1	(R)	7	85	15
16.0 mm.	Carnegie 5542B	6.2.4	(R)	8	85	40
20.0 mm.	Carnegie 462	15.2.1	(R)	9	85	40
22.0 mm.	Carnegie 4960	23.2.3	(L)	10	85	15
25.0 mm.	Wisconsin 87	23.1.6	(L)	11	85	10
27.5 mm.	Carnegie 2561	31.3.2	(L)	12	85	25
30.0 mm.	Wisconsin 92	34.2.2	(R)	13	85	10
30.0 mm.	Wisconsin 92	33.2.4	(L)	14	85	10
30.0 mm.	Wisconsin 92	33.1.2	(L)	15	85	10
30.0 mm.	Wisconsin 92	33.1.2	(L)	16	300	10
34.0 mm.	Carnegie 6647	27.1.3	(R)	17	85	30
34.0 mm.	Carnegie 6647	27.1.3	(R)	18	300	30
43.0 mm.	Wisconsin 90	8.1.1	(L)	19	85	15
50.0 mm.	Wisconsin 86	20.1.8	(L)	20	85	15
50.0 mm.	Wisconsin 86	19.5	(L)	21	85	15
50.0 mm.	Wisconsin 86	19.5	(L)	22	300	15

All the embryonal and fetal series (Carnegie and Wisconsin) were sectioned in transverse planes; all measurements were in crown-rump length.

Reconstructions were prepared from the following series:

25 mm. (eight weeks)	Wisconsin 87
50 mm. (eleven weeks)	Wisconsin 86

The dimensions presented in the text pertaining to the 25 mm. and 50 mm. series were derived from the reconstructions; all other dimensions are based on computations of section thicknesses.

HISTORICAL REVIEW

The anatomic development of the stapes has been the subject of microscopic study for more than a century, a fact which suggests that the conclusions reached by the earlier investigators were unsatisfactory to the later ones. The various opinions warrant consideration at this time.

Reviews of the literature on stapedial development have been presented by Fraser[2] (1882), Gradenigo[3] (1887), Dreyfuss[4] (1893), Broman[5] (1899), Kingsley[6] (1900), Fuchs[7] (1905), Reagan[8] (1917),

2. Fraser, A.: On the Development of the Ossicula Auditus in the Higher Mammalia, Phil. Tr. Roy. Soc., London **173** (pt. 2):901-926, 1882.

3. Gradenigo, G.: Die embryonale Anlage des Mittelohres; die morphologische Bedeutung der Gehörknöchelchen, Med. Jahrb. **83**:61-120 and 219-308, 1887.

4. Dreyfuss, R.: Beiträge zur Entwickelung des Mittelohres und des Trommelfells des Menschen und der Saugethiere, Morphol. Arb. **2**:607-662, 1893.

5. Broman, J.: (*a*) Ueber die Entwickelung der Gehörknöchelchen beim Menschen, Verhandl. d. anat. Gesellsch. **12**:230-236, 1898; (*b*) Die Entwickelungsgeschichte der Gehörknöchelchen beim Menschen, Anat. Hefte **11**:509-670, 1899.

6. Kingsley, J. S.: The Ossicula Auditus, Tufts College Series, Boston, 1900, no. 6, pp. 203-274.

and van der Klaauw [9] (1924); the last-named author includes as well an exhaustive compilation of the literature on the comparative anatomic development of the auditory apparatus.

Investigators have derived the stapes from (1) the first visceral (mandibular) arch or bar, (2) the second visceral (hyoid) arch or bar, (3) the otic capsule, (4) the otic capsule and the second visceral arch, and (5) a "periarterial" aggregate of cells distinct from visceral or cranial elements.

Valentin [10] (1835), Guenther [11] (1842) and Coyle [12] (1909) maintained that the stapes originates from the mandibular arch. Guenther described the division of the first postoral cartilage into three parts, from the medial element of which arose a nodule so closely applied to the labyrinth that it produced a depression at the future site of the vestibular (oval) window. On this nodule, in turn, according to Guenther, an extension developed which became bent on itself to form the stapes and the long process of the incus. Coyle, studying a series of embryos of the horse representing stages from three weeks to term, distinguished between the visceral arch and the visceral bar (following the lead of Reichert, and later, of Fuchs) and concluded that the ossicular elements (including the stapes) generally arose from the first visceral arch but that the malleus was exceptional in being a derivative of the corresponding visceral bar (Meckel's).

Reichert [13] (1837) first ascribed the total origin of the stapes to the second visceral (hyoid) bar. This bar was described as being attached

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7. Fuchs, H.: (*a*) Bemerkungen über die Herkunft und Entwickelung der Gehörknöchelchen bei Kaninchen-Embryonen (nebst Bemerkungen über die Entwickelung des Knorpelskeletes der beiden ersten Visceralbogen), Arch. f. Anat. u. Entwcklngsgesch., 1905, supp., pp. 1-178; (*b*) Untersuchungen über die Entwickelung der Gehörknöchelchen, des Squamosums und des Kiefergelenkes der Säugetiere, nebst einigen vergleichendanatomischen Betrachtungen über Articulare, Quadratum und Gehörknöchelchen, ibid., 1906, supp., pp. 1-90.

8. Reagan, F. D.: The Role of the Auditory Sensory Epithelium in the Formation of the Stapedial Plate, J. Exper. Zool. **23**:85-108, 1917.

9. van der Klaauw, C. J.: Bau und Entwicklungsgeschichte der Gehörknöchelchen, Ergebn. d. Anat. u. Entwcklngsgesch. **25**:565-622, 1924.

10. Valentin, G.: Handbuch der Entwickelungsgeschichte des Menschen, Berlin, A. Rücker, 1835, p. 97.

11. Guenther, A. F.: Beobachtungen über die Entwickelung des Gehörorgans, bei Menschen und höheren Säugethiere, Leipzig, Wilhelm Engelmann, 1842.

12. Coyle, R. F.: The Development of the Auditory Ossicles in the Horse, with a Note on Their Possible Homologies in the Lower Vertebrates, Proc. Roy. Soc. Edinburgh **29**:582-600, 1909.

13. Reichert, C.: Ueber die Visceralbogen der Wirbelthiere im Allgemeinere, und deren Metamorphosen bei den Vögeln und Säugethieren, Arch. f. Anat., Physiol. u. wissensch. Med., 1837, pp. 120-222.

originally to the chondrocranium in the region of the postsphenoid; with disappearance of this part, the remainder of the bar formed the stapes proximally and, in succession, the stapedial muscle, the pyramidal eminence, the styloid process, the stylohyoid ligament and the lesser cornu of the hyoid bone. Several later writers were protagonists of this view: Gadow[14] (1888), Gaupp[15] (1899) and Kingsley[6] (1900) approaching the problem from the phylogenetic standpoint, based their conclusions on gross dissections of comparative vertebrate material. According to Gadow, the whole mammalian ossicular chain represents a modification of the hyomandibula, which is the primitive proximal derivative of the second visceral arch; Gaupp and Kingsley supported the main opinion of Gadow but favored the view that the incus is homologous to the quadrate, the malleus to the articular bone. Wiederscheim[16] (1907) simply stated that the stapes corresponds to the upper element of the hyoid arch (the piscine hyomandibula) and that the intimate relationship between the stapes and the otic capsule is probably a secondary condition. Rabl[17] (1887) and later Hertwig[18] (1892) found collateral proof of the origin of the stapes from the hyoid arch in the fact that the stapedial muscle is innervated by the facial nerve (of the second arch) and that the stapes is perforated by a branch of the internal carotid artery.

Microscopic studies contributed information of a more direct character. In 1892 Baumgarten,[19] studying a single 30 cm. human fetus, commented on the difficulty of differentiating the primordium of the stapes from the adjacent embryonic connective tissue of the labyrinth (lamina stapedialis), but in considering it highly probable that the hyoidal cartilage alone participated in the formation of the stapes he incidentally ruled out the possibility that the base of the stapes might arise in part from the connective tissue lamina. Zondek[20] (1895) identified an uninterrupted connection between the *Bildungsmasse* (for-

14. Gadow, H.: On the Modification of the First and Second Visceral Arches with Special Reference to the Homologies of the Auditory Ossicles, Phil. Tr. Roy. Soc., London, s.B **179**:451-486, 1888.

15. Gaupp, E.: Ontogenese und Phylogenese des schallbeitenden Apparates bei den Wirbeltieren, Ergebn. d. Anat. u. Entwcklngsgesch. **8**:990-1149, 1899.

16. Wiederscheim, R.: Comparative Anatomy of Vertebrates, translated by W. N. Parker from the sixth German edition, ed. 3, London, Macmillan & Co., 1907.

17. Rabl, K.: Ueber das Gebiet des Nervus facialis, Anat. Anz. **2**:219-227, 1887.

18. Hertwig, O.: Textbook of the Embryology of Man and Mammals, translated from the third German edition by E. L. Mark, New York, Macmillan & Co., 1892.

19. Baumgarten, H.: Beiträge zur Entwicklungsgeschichte der Gehörknöchelchen, Arch. f. mikr. Anat. **40**:512-530, 1892.

20. Zondek, M.: Beiträge zur Entwicklungsgeschichte der Gehörknöchelchen, Arch. f. mikr. Anat. **41**:499-509, 1895.

mation mass) of the hyoid arch and the primordial tissue of the stapes. Broman [5b] maintained unequivocally that the stapes arose from the hyoid arch and from it alone. Broman observed perforation of the blastema of the stapes by the stapedial artery in the earliest stage of development; he termed "interhyal" the connection between the stapes and the hyoid arch and recognized that the structure never differentiated beyond the stage of dense embryonic connective tissue. Hegetschweiler [21] (1898) likewise expressed the belief that the stapes was derived from the hyoid bar but, like Baumgarten and Zondek, tended to be equivocal in his opinion regarding the importance of the lamina stapedialis. The footplate (base) was not considered to be of labyrinthine origin because the lamina, though derived from the precartilaginous capsule, not only failed to differentiate beyond that stage of cellular evolution but actually underwent gradual dedifferentiation into connective tissue. Early connection between the stapedial blastema and the hyoid cartilage was observed by Eschweiler [22] (1911) in a series of pig embryos. He found that this "precartilaginous" bridge disappeared soon after the 30 mm. stage. From its inception the stapes was clearly separated from the blastema of the labyrinth by a pale-staining mesenchymal tissue, the so-called intermediate zone.

Opposed to the view that identifies the stapes as a derivative of the first or of the second arch or bar is the contention held by several investigators that the stapes arises from the otic capsule itself. Burdach [23] (1828) was one of the earliest investigators to commit themselves to this view. Parker [24a] (1874) and later Parker and Bettany [24b] (1877) expressed the belief that the stapes arose from a median lobular protuberance on the outer surface of the otic capsule; this mass eventually freed itself from the wall of the labyrinth but seemingly remained as a plug in the future vestibular window. Gruber [25] (1877) contended not only that the stapes arose from the primordial skeleton of the embryonic head and therefore from the same structural material (mesenchyma) that gave rise to the labyrinthine capsule but that the

21. Hegetschweiler, J.: Die embryologische Entwicklung des Steigbügels, Arch. f. Anat. u. Entwcklngsgesch., 1898, pp. 37-56.

22. Eschweiler, R.: Zur Entwicklungsgeschichte des M. Stapedius und des Stapes, Arch. f. mikr. Anat. **77**:52-77, 1911.

23. Burdach, C. F.: Die Physiologie als Erfahrungswissenschaft, Leipzig, L. Voss, 1828, vol. 2, pp. 456 and 465; cited by Fraser.[2]

24. (*a*) Parker, W. K.: On the Structure and Development of the Skull in the Pig (Sus Scrofa), Phil. Tr. Roy. Soc., London **164**:289-336, 1874. (*b*) Parker, W. K., and Bettany, G. T.: The Morphology of the Skull, London, Macmillan & Co., 1877.

25. Gruber, J.: (*a*) Beitrag zur Entwickelungsgeschichte des Steigbügels und ovalen Fensters, Monatschr. f. Ohrenh. **11**:154-158, 1877; (*b*) Zur Entwickelungsgeschichte des Säugethiere und des Menschen, ibid. **12**:54, 1878.

malleus and the incus were likewise derived from the capsular tissue. The concept of a purely labyrinthine origin attracted little serious attention, however, until the appearance of Fuchs's [7] voluminous monographs in 1905 and 1906, based on a study of rabbit embryos. Fuchs stated that the stapedial blastema originally belonged histologically and topographically to the otic capsule. The anlage of the stapes appeared wholly independent of the anlages of the first and second visceral structures.[26] The stapedial blastema, then, was a derivative of the mesenchymal *Skeletanlage* (skeletal anlage) in the region of the labyrinth and of the precartilage comprising the capsule itself. In Fuchs's opinion further differentiation of the precartilaginous stapes from the cytoblastema resulted in the investiture of the adjacent stapedial artery. Continued lateral growth of the stapes effected a connection with the precartilaginous tissue of the second visceral arch; this was described as an entirely secondary union, maintained temporarily by the so-called hyostapedial ligament. The later definitive stapes was derived, therefore, from the stapedial ring of cartilage.[27]

A composite origin entailing stapedial derivation partly from the second visceral bar and partly from the otic capsule was supported by Gradenigo [3] (1887), van Noorden [28] (1887), Hertwig [18] (1905), Jenkinson [29] (1911), Macklin [30] (1914), Reagan [8] (1917) and McClain [31] (1939).

From a study of feline, porcine, canine, murine and human embryos, Gradenigo concluded that the stapedial ring arose from the second visceral bar, and the major part of the basal plate from the differentiating lamina stapedialis of labyrinthine origin. This view agrees with that of Hegetschweiler except that the latter stressed the qualitative factor in the laminar contribution to the footplate; he considered

26. The anlage of the stapes lies in the so-called paralabyrinthine space, situated medial to the labyrinthine anlage, lateral to the dorsal segment of the first pharyngeal pouch and ventral to the posterior wall of the upper part of the foregut.

27. This concept of stapedial origin considered from the standpoint of phylogeny would make untenable the theory of possible homology between the columella-extracolumella and the hyomandibula (originating from the second visceral arch) of Sauropsida and Fishes, respectively. Since Fuchs [7b] derived the mammalian stapes from the otic capsule alone, he defined that structure as a homologue of the amphibian operculum.

28. van Noorden, W.: Beitrag zur Anatomie der knorpeligen Schädelbasis menschlichen Embryonen, Arch. f. Anat. u. Entwcklngsgesch., 1887, pp. 241-257.

29. Jenkinson, J. W.: The Development of the Ear Bones in the Mouse, J. Anat. & Physiol. **45**:305-318 (July) 1911.

30. Macklin, C. C.: (*a*) The Skull of a Human Fetus of 40 mm., Am. J. Anat. **16**:317-426, 1914; (*b*) The Skull of a Human Fetus of 43 mm. Greatest Length, Contrib. Embryol. **10**:59-103, 1921.

31. McClain, J. A.: The Development of the Auditory Ossicles of the Opossum (Didelphys Virginiana), J. Morphol. **64**:211-266, 1939.

only the vestibular surface of the base to be of laminar derivation. Van Noorden described an extramural contribution from the second visceral bar, represented by the head and the crura, and an intramural portion from the fenestral tissue forming the basal mass. A true footplate was not seen, owing to the early age of the specimens.

Jenkinson's work on embryos of mice corroborated the view that the stapes is derived from the dorsal median portion of the hyoid bar. The stapedial blastema was found to be perforated at the earliest stage by the stapedial artery; then, assuming a rounded form, it became separated from the remainder of the hyoid. Originally distinct from the capsular blastema, the stapes became embedded in the capsule secondarily, and the tissue separating the stapes from the vestibule underwent dedifferentiation from precartilaginous into fibrous connective tissue. Agreeing with Gradenigo, the author considered the lamina stapedialis as contributing to the stapedial structure but insisted on a direct labyrinthine origin.

On reconstruction of the skull of a human fetus of 40 mm., Macklin described "the elongated crescentic fenestra vestibuli, lying in a general direction from above downward and backward and presenting a concavity downward and forward." The concavity contained the anlage of the footplate of the stapes, which filled only a small portion of the space, the remainder being occupied by the connective tissue representative of the annular ligament of the base. The latter "anlage" was the lamina stapedialis (see hereinafter).

Employing experimental methods, Reagan offered a striking demonstration that in birds the homologue of the stapes is apparently of mixed origin, cranial and visceral. In cases in which the otocyst had been removed from the embryo, a "stapedial" basal rim failed to form, while the columellar portion of the stapes attained full length and normal proportions. It appeared, therefore, that the central portion of the base, together with the columellar part, was derived from the second visceral bar; the "flange" or basal rim, on the other hand, arose from the fenestral margin (capsular) by independent chondrification. McClain recently contributed information on Didelphys that is in close agreement with Reagan's experimental findings on the chick. The peripheral flange of the stapes was found to be derived from the tissue of the otic capsule that also formed the articular rim of the vestibular window and the annular ligament. He based his conclusions on tinctorial methods of cellular differentiation and utilized only those stages in which the footplate had already formed. The cells forming the flange in the 20 to 25 day pouch fetus are described as developmentally and tinctorially identical with those forming the fenestral rim, while the intervening annular ligament is a well defined structure.

Visceral and somatic skeletal elements take no part whatsoever in the development of the stapes as viewed by Magitot and Robin [32] (1862), Hunt [33] (1877), Salensky [34] (1880), Fraser [2] (1882), Staderini [35] (1891), Dreyfuss [4] (1893) and Siebenmann [36] (1894). Magitot and Robin offered a vague description of an independent mesenchymal origin of the incus and the stapes. Hunt stated that the stapes probably arose in embryonic connective tissue, like the other ossicles. Salensky's observations on embryos of sheep and pigs indicated that the stapes arose independently of the other ossicles and the otic capsule, having its inception as a clump of mesenchymal cells surrounding the mandibular branch (stapedial) of the internal carotid artery. He pointed out, as had Reichert, Jenkinson and others, that the blastema was occupied by an artery during its earliest stages of development rather than perforated through secondary rearrangement (Fuchs, 1905). Fraser similarly described the developing stapes as a circular mass of cells surrounding the stapedial artery, and to that vessel he delegated an important role in the initial formation and future development of the ossicle. While the stapes was stated to be histologically similar to the adjacent incus and otic capsule, it was considered to be of strictly independent origin, any contact with the hyoid bar or the otic capsule being an entirely secondary feature. Staderini concluded that the primordial stapedial ring developed independently of the proximal ends of both visceral bars, but he was only partly in accord with Salensky's idea of a "periarterial" origin: The stapedial blastema arose in the vicinity of the labyrinth from embryonic mesenchyma and was then secondarily invaded by a branch of the internal carotid artery. Dreyfuss' observations on embryos of porpoises, sheep, rabbits, mice and man indicated that the stapes originated free in embryonic connective tissue and became secondarily associated with the otic capsule. The stapedial ring was recognized early as a concentrically layered cell mass lying between the blastemas of the proximal ends of the first and second visceral bars; attachment to either bar was likewise a secondary step. The definitive stapes was

32. Magitot, E., and Robin, C.: Mémoires sur un organe transitiore de la vie foetale désigné sous le nom de cartilage de Meckel, Ann. d. sc. nat. **18**:213, 1862.

33. Hunt, D.: On the Development of the External Ear Passages, Am. J. M. Sc. **73**:58-62, 1877.

34. Salensky, W.: Beiträge zur Entwickelungsgeschichte der Knorpeligen Gehörknöchelchen bei Säugethieren, Morphol. Jahrb. **6**:415-432, 1880.

35. Staderini, R.: Intorno alle prime fasi di suillupo dell' annulus stapedialis, Monitore zool. ital. **2**:147, 1891.

36. Siebenmann, F.: Die ersten Anlagen von Mittelohrraum und Gehörknöchelchen des menschlichen Embryo in der 4. bis 6. woche, Arch. f. Anat. u. Entwcklngsgesch., 1894, pp. 355-365.

derived, nevertheless, only from the original annulus. In the following year (1894) Siebenmann found from a study of human embryos ranging in length from 7 mm. to 15.5 mm. (4 to 6 weeks) that the stapedial blastema was observable in the 7 mm. embryo, located on the dorsal margin of the first visceral pouch and ventrolateral to the labyrinthine anlage but altogether independent of that structure and the ventrally placed first and second visceral bars. He was convinced that the controversy over the visceral or the cranial origin of the stapes was entirely superfluous, and he maintained the independence of all cartilaginous anlages in the neighborhood of the capsule. As Keibel [37] pointed out, however, Siebenmann's opinion must be considered lightly because of his disregard for the interpretative importance of comparative anatomy and the basic developmental pattern of the mammalian skull.

OBSERVATIONS AND COMMENT

6.7 Mm. Embryo (Carnegie 6508).—In the 6.7 mm. embryo the hyoid bar is identifiable as a circumscribed condensation of mesenchymal cells located within the second (hyoid) branchial arch; cranially it is situated on the lateral aspect of the facial nerve; it turns medially at the caudal portion of the otocyst, where it is lodged between the nerve and the first visceral cleft. It terminates abruptly at this level; its bluntly rounded end gives no evidence of association with a stapedial primordium. There is no condensation of mesenchyma in the immediate vicinity of the otocyst that might suggest the formation of an otic capsule; moreover, there is none within the first visceral arch at this level to forecast the position of Meckel's bar.

7 Mm. Embryo (Carnegie 617).—In the 7 mm. stage the second visceral hyoid bar projects cranialward (as in the preceding stage) and turns medially between the facial nerve and the first visceral cleft to form a bulbous mesenchymal mass on the medial aspect of the facial nerve. This mass, the primordium of the stapes, maintains cellular continuity with the hyoid bar; it is located lateral and somewhat caudal to the otocyst but is wholly separate from the otocyst and from the meager condensation of mesenchymal cells closely investing the periphery of that structure. This is the earliest stage in man in which a definite stapedial cytoblastema could be recognized. Clearly, then, the human stapes arises as a solid mass of mesenchyma at or from the medial and the cranial end of the second visceral (hyoid) bar. A stapedial artery is not yet present, but at a lower level a bifid branchial artery passes dorsolaterally into the medial aspect of the second branchial arch, and

37. Keibel, F., and Mall, F. P.: Manual of Human Embryology, Philadelphia, J. B. Lippincott Company, 1912, vol. 2.

it seems probable that this vessel will soon encroach on and penetrate the now solid blastema. Meckel's bar has not yet appeared at this level.[38]

8 Mm. Embryo (Carnegie 559).—The cytoblastema of the stapes in the 8 mm. embryo is a slightly irregular ovoid mass of condensed mesenchymal cells penetrated for the first time by a small vessel which represents the primitive stapedial branch of the second branchial (arterial) arch. The cranial portion of the stapedial blastema is still completely separate from the otic capsule—the latter being identifiable as a mesenchymal condensation along the posterolateral aspect of the otocyst. The bridge between the stapes and the hyoid bar produces a concave impression on the under surface of the facial nerve just distal to the geniculate ganglion (as the facial nerve begins its posteroinferior course), so that a section through the midportion of the stapes reveals the following topographic relations: The stapes lies inferolateral to the otic capsule, between the geniculate ganglion anteriorly and the descending portion of the facial nerve posteriorly; the hyostapedial bridge appears immediately posterior to the ventral limb of the anterior cardinal vein and anterolateral to the facial nerve proper. Caudal to the lowermost portion of the stapedial blastema, the hyoid bar lies to the medial aspect of the facial nerve.[39]

9 Mm. Embryo (Carnegie 887).—This specimen closely resembles the preceding one. The stapes is lodged within the tissue of the second branchial arch, continuous with the caudal extremity of the corresponding visceral bar. The vessel which is the source of the stapedial artery (the second branchial artery) courses deep to the otic capsule and immediately medial to the stapedial blastema; the proximal portion of the interstapedial artery is bifid, the two tiny twigs becoming confluent as the lateral aspect of the cytoblastema is approached.

9.6 Mm. Embryo (Carnegie 6517).—In the 9.6 mm. embryo the blastema of the stapes, consisting of a dense aggregate of mesenchymal cells, appears immediately lateral to the capsule and medial to the facial nerve (figs. 1 and 2). The otic capsule consists of condensed

38. This specimen was sectioned symmetrically, so that both sides were represented at almost precisely the same level in any given section. Consequently the relative positions of the otocyst and the stapedial blastema could be accurately ascertained; the latter structure does not make its appearance (passing caudally) until the inferior extremity of the otocyst has been reached. The stapes therefore approaches the inferolateral aspect of the otic capsule; the significance of this observation will become apparent in the light of the interpretation of obliquely sectioned specimens.

39. The term "Reichert's cartilage," when used, is applied specifically to that portion of the hyoid bar distal to the tympanohyal (primordium of the styloid base); from it are derived the styloid process proper, the stylohyoid ligament and the lesser cornu of the hyoid bone.

mesenchyma. The periotic mesenchyma, less dense than that of the stapes, is distinguishable from the stapes by a zone of demarcation.

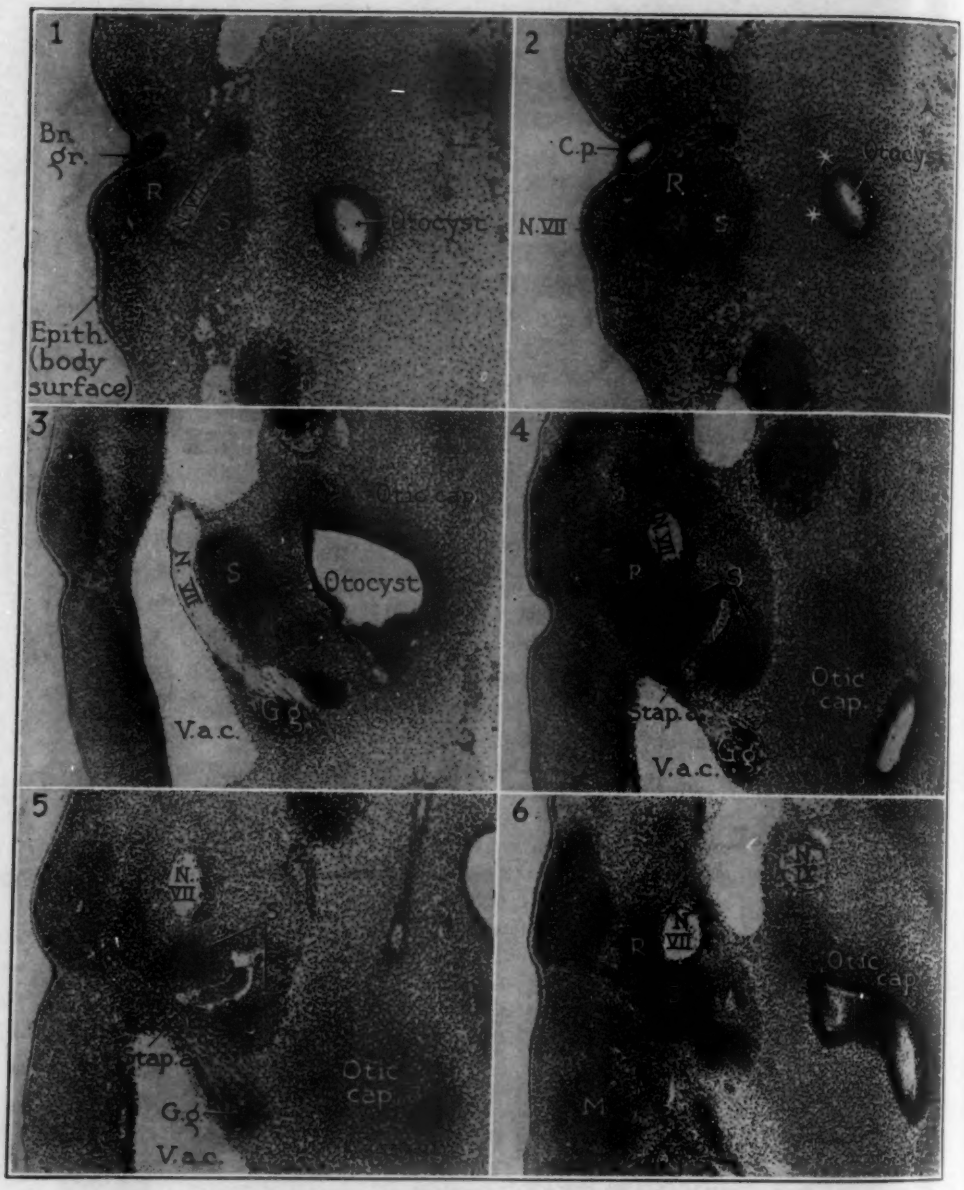
The only tissue in the vicinity comparable in structure and density to the stapedial blastema is that of the second visceral (hyoid) bar; it lies lateral to the facial nerve, at the level of the stapes. These two masses are continuous 65 to 70 microns caudal to the cranial extremity of the stapes (fig. 2).

The stapedial blastema is not perforated by a distinct arterial twig in this particular specimen. A stapedial artery occurs in the 8 mm. embryo and in the 9 mm. specimen; its absence here perhaps represents retardation in vascular development, entirely within the normal range of ontogenetic variation; all succeeding series were characterized by the presence of a stapedial vessel, but that structure too was subject to variation in its size and course.

10.6 Mm. Embryo (Carnegie 6524).—At this stage the stapedial and capsular concentrations of cells are closely approximated (figs. 3 and 4). Yet here, as in all specimens, regardless of age, the blastema of the stapes can be readily distinguished from the adjacent capsular tissue (whether condensed mesenchyma, precartilage or true cartilage) by the presence of a pale-staining limiting zone of mesenchyma consisting either of loose reticulum or more densely arranged, radially directed mesenchymal cells.[40]

In the 10.6 mm. embryo the stapes is penetrated by a relatively large stapedial artery, the proximal (medial) portion of which is widely dilated. Its source and termination could not be accurately determined.

40. Because of the oblique character of the sections in this series, the cranial extremity of the stapedial blastema appears as a small rounded mass embedded in the substance of the periotic mesenchyme (compare Fraser[2]). Casual observation suggests an intracapsular origin of the stapes with secondary extrusion into the tympanic cavity, but closer examination reveals that a definite collar of pale-staining mesenchyme separates the deeply staining stapedial mass from the periotic mesenchymal tissue. Caudalward in the series the stapes is found on the lateral periphery of the capsule, and as the level of junction of the stapes and the hyoid bar is reached in succeeding sections the stapedial mass is clearly separated from the periotic tissue. The blastema of the stapes at its inception is located at the inferolateral aspect of the otic capsule; by concomitant growth these two structures gradually become more closely approximated until the cranial extremity of the stapes actually impinges on the pale-staining fibrillar mesenchyma at the capsular periphery. In all obliquely sectioned series the stapedial cytoblastema appears to arise within the otic capsule itself, but the early appearance of the stapes as a distinct outgrowth of a preformed visceral skeletal element, its marked distance from the otocyst and the denser aggregation of mesenchyma cells forming its mass in comparison with the loose reticular nature of the early periotic mesenchyma point to the incontrovertible fact that the intimate relationship of the stapes and the capsule in somewhat later stages is an entirely secondary one.



Figures 1 to 6

(See legend on opposite page)

11 Mm. Embryo (Carnegie 1836).—The most arresting otologic feature of this series is the circumneural arrangement of the blastema of the stapedial ring and the hyoid bar at the caudal extremity of the otic capsule; continuity between these two structures was observed not only at the usual site (i. e., ventral to the facial nerve) but also cranially, where dorsal prolongations of the stapes and the visceral bar effect a secondary fusion. The preneural and postneural portions of these adjacent structures therefore form a ring of mesenchymal tissue joining the stapes and the hyoid bar.

The stapedial blastema is perforated by an artery of relatively large caliber.

12 Mm. Embryo (Carnegie 6521).—The stapedial blastema itself and the stapedial extremity of the hyoid bar are unusually massive in this specimen; the craniocaudal dimension of the stapes (estimated) is 0.56 mm., and continuity with the visceral arch is maintained almost throughout its extent.

The stapes and the otic capsule are intimately related near the cranial extremity of the former, but caudally this intimacy is gradually

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EXPLANATION OF FIGURES 1 TO 6

Sections through the stapedial region in embryos 9.6 mm. to 13.1 mm. in length (figs. 1 and 2, 9.6 mm.; figs. 3 and 4, 10.6 mm.; fig. 5, 12.9 mm.; fig. 6, 13.1 mm.); × 26. Figure 1 shows the stapedial blastema developing at a distance from the loose mesenchymal tissue surrounding the otocyst. Figure 2, through the lower third of the otocyst, shows the interval between the stapes and the hyoid bar ventral to the facial nerve. The asterisks mark the primordial otic capsule. Figure 3 shows the cranial portion of the dense stapedial blastema approaching the otocyst; the auditory structures are related laterally to the anterior cardinal vein. Figure 4 shows the middle of the stapes, with the stapedial artery and the more definite connection between the stapes and the otic capsule. Figure 5 shows the stapes with a branching stapedial artery. Figure 6 reveals the persistent connection between the stapes and the hyoid bar and shows Meckel's (hyoid) bar at the level of the lower fourth of the otocyst.

In these and succeeding figures *Ang. m.* indicates the angular (canalicular) mass; *Ann. lig.*, the annular ligament; *Br. gr.*, the first branchial groove; *C.* or *Coch.*, the cochlea; *Ca. c.*, the canalicular cartilage; *Co. c.*, the cochlear cartilage; *C. p.*, the closing plate (of branchial groove); *Epith.*, the surface epithelium; *F. a. f.*, the fissula ante fenestram; *Fen. rim.*, the fenestral rim; *G. g.*, the geniculate ganglion; *I*, the incus; *Lam. stap.*, the lamina stapedialis; *M*, Meckel's bar; *N. VII*, the facial nerve; *N. IX*, the glossopharyngeal nerve; *Otic cap.*, the otic capsule; *R*, the hyoid (Reichert's) bar; *S*, the stapes; *S. c.*, the semicircular canal; *Stap. a.*, the stapedial artery; *T. c.*, the tympanic cavity; *T. t. m.*, the tensor tympani muscle; *Tymp. mesen.*, the tympanic mesenchyma; *V.* or *Vest.*, the vestibule; *V. a. c.*, the anterior cardinal vein; *V. p.* or *Visc. p. 1*, the first visceral pouch (primordium of auditory tube).

lost. Histologic and tinctorial distinctions between the component cellular elements in these two structures have been so constantly demonstrated in successive series that the notion of an origin of the stapes blastema in the primary capsule (Fuchs, 1905) is discredited.

The stapedial artery is not a constant single vessel. The left stapes in this embryo is penetrated by a relatively large artery (0.03 mm. in diameter) which maintains a direct intrablastemal course; the right stapes, on the other hand, is canalized by a veritable rete. The main vessel enters the medial side of the mesenchymal mass as a single twig and immediately widens and subdivides into several irregular branches, two of which finally emerge from the lateral aspect of the stapes.

12.9 Mm. Embryo (Carnegie 6520).—The general topography of the stapedial and capsular regions is very similar to that described for the preceding stage. However, the stapedial blastema consists of dense mesenchyma of a character comparable only to the periotic condensation along the medial and caudal aspects of the otic cavity; the remainder of the capsule consists of somewhat less compact pale-staining mesenchyma, blending imperceptibly with the loose reticulum at the periphery. Even at the cranial extremity of the stapes, where this structure presses closely on the capsular tissue, a zone of demarcation between the two structures is invariably discernible.

The right stapedial artery resembles that of the same side in the preceding (12 mm.) specimen. It is greatly dilated as it approaches the medial aspect of the stapedial blastema; within the mass it breaks up into three small distinct channels, but these on emergence are brought together by anastomosis in the mesenchyma of the second arch (the two cranialmost channels in fig. 5).

13.1 Mm. Embryo (Carnegie 6527).—In the 13.1 mm. embryo the otic capsule is undergoing rapid increase in size—a much greater relative increase than that experienced by the stapes. Histologic and tinctorial distinctions are marked (as in the 12.9 mm. embryo), it being clear that the periotic mesenchyma comparable to the stapedial blastema is chiefly limited to the medial aspect of the otic cavity while the line of stapediocapsular apposition is clearly defined (fig. 6).

The attachment of the stapes to the hyoid bar is still maintained (fig. 6), but only at the inferolateral portion of the stapes—which is the part destined to become the capital or lateral extremity of the ossicle.

A ventrally directed branch of the stapedial artery, after emergence from the stapes, extends into the medial portion of the first branchial arch to approach the first visceral pouch.

13.95 Mm. Embryo (Carnegie 4430).[41]—In embryos beginning with the 8.0 mm. stage (excepting that at the 9.6 mm. stage) the blastema of the stapes is penetrated by one or more vascular channels representing the stapedial artery, and although the axial character of the vessel is readily seen in the 10.6 mm and 13.1 mm. specimens, a well defined annular stapes, possessing a histologically distinct obtutrator margin and separated from the stapedial artery by an adventitia of loose mesenchyma, is first demonstrated at this stage of development.

As a result of concomitant growth, the stapes and the capsule are in close apposition. The hyostapedial attachment is still strong (fig. 7) but is now only 60 microns in thickness.[42]

15 Mm. Embryo (Carnegie 719).—The stapes is now definitely annular in form, and the crura are circular in cross section. The dorsal crus is estimated to be about one-half the diameter of the ventral crus. The dorsolateral portion of the ring effects connection with the hyoid bar; this junction occurs caudal to the bridge of the facial nerve, as hereinbefore described for other stages.

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41. Technical imperfections detract from the value of a preceding stage, a 13.5 mm. embryo (Carnegie 695). The mesenchymal structures at and below the stapedial level are indistinct, but multiple interstapedial branches of the stapedial artery can be observed on the left side. In a succeeding stage, a 14 mm. embryo (Carnegie 6258), the stapes is transfixed by an artery of relatively large size, but again cellular distinctions are not clear at the caudal stapedial and capsular levels.

42. It should be recorded that in this series the os lentiforme seems to be derived not from the distal end of the processus longus but from mesenchymal tissue lying immediately adjacent to the stapes. Thus in section 11.2.3, near the caudal extent of the hyostapedial attachment, the prominent incudal portion of Meckel's bar approaches the inferolateral aspect of the stapes. Between the distal extremity of the incus and the adjacent stapes lies a small mesenchymal mass quite separate from either of the aforementioned structures. In the following section (11.2.4.) the apparently isolated mass blends with the dense surface mesenchyma of the lateral portion of the stapes and is still demarcated from the incus by a narrow strip of pale-staining tissue. Kingsley[6] and L. Drüner (Ueber die Anatomie und Entwicklungsgeschichte des Mittelohres beim Menschen und bei der Maus, Anat. Anz. **24**:257, 1904) derived the os lentiforme from the processus longus during its mesenchymal stage; C. S. Mead (The Chondrocranium of an Embryo Pig, Sus Scrofa: A Contribution to the Morphology of the Mammalian Skull, Am. J. Anat. **9**:167, 1909), from the processus longus during the precartilaginous stage. On the other hand, A. Magnus (Die Hebelbewegungen des Hammers und des Amboss, Virchows Arch. f. path. Anat. **20**:79, 1861) and E. Kallius (Beiträge zur Entwicklung der Zunge: 3. Teil; Säugetiere 1; Sus scropha dom., Anat. Hefte **41**:173, 1910) claimed for it a separate origin from the thick connective tissue which binds the stapes and the incus, and secondary fusion with the distal extremity of the processus longus.

Again, in the 17 mm. embryo the distal articular end of the incus appears at the same level as the body of the long process but separated from it by a hiatus; however, passing caudally the distal and proximal portions become continuous, indicating a curved incudal terminus adjacent to the stapes.

Figures 7 to 12

(See legend on opposite page)

Meckel's bar is situated caudal to the stapes; the hyoid bar appears dorsomedial to the first visceral pouch and medial to the chorda tympani of the facial nerve.

The stapedial artery is prominent.

15.5 Mm. Embryo (Carnegie 2673).—Developmentally this specimen closely resembles the preceding (15 mm.) one. The hyostapedial attachment is about 25 microns in thickness. The distal end of the incus closely approaches but does not actually touch the stapes; the isolated mesenchymal mass tentatively identified as the os lentiforme (processus lenticularis) of the incus in the 13.95 mm. embryo is not apparent in this or in the 14 mm. and 15 mm. specimens.

15.8 Mm. Embryo (Carnegie 4405).—While both the otic capsule and the stapes are engaged in rapid dimensional expansion, the former structure demonstrates at this stage a perceptibly greater increment than the latter. At the 9.6 mm. stage the stapes was 0.195 mm. in its greatest linear dimension, while at the 15.8 mm. stage there is an increase of only 0.065 mm. The epithelium-lined otic cavity, on the other hand, has greatly enlarged and has acquired a considerable periotic accumulation of mesenchyma, the central portion of which shows transition into precartilage. Dorsolateral extension of the periotic tissue gives rise to the earliest indication of the so-called angular mass—that portion of the capsule destined to house the semicircular canals.

On its craniomedial aspect the stapes is closely applied to the lateral capsule immediately ventral to the angular mass; the capsular mesen-

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EXPLANATION OF FIGURES 7 TO 12

Transverse sections through the stapedial and capsular regions in embryos 13.95 mm. to 27.5 mm. in length (fig. 7, 13.95 mm.; fig. 8, 16 mm.; fig. 9, 20 mm.; fig. 10, 22 mm.; fig. 11, 25 mm.; fig. 12, 27.5 mm.); × 26. Figure 7 shows the mesenchymal attachment between the stapes and the hyoid bar; the stapes is lateral to and separate from the otic capsule. Meckel's bar lies in the first visceral arch. Figure 8 shows the persisting hyostapedial attachment; also, a distinctly annular stapes with its axial stapedial artery. Figure 9 shows approximation of the long process of the incus to the lateral extremity of the stapes. The stapes is in a transitorial histologic stage between dense mesenchyma and precartilage. Figure 10 shows the precartilaginous stapes in close contact with the lateral capsular wall. The hyostapedial attachment (future stapedius tendon and muscle), at the asterisk, is attached to the angular portion of the canalicular cartilage mass. Figure 11 shows the distinctly precartilaginous stapes in juxtaposition to a correspondingly precartilaginous lamina stapedialis (between tips of arrow). The malleus, the incus, and the angular mass are composed of young true cartilage. Figure 12 shows the stapes in transition between precartilage and cartilage. The stapes lies adjacent to the precartilaginous lamina stapedialis (latter marked by asterisk).

chyma (early precartilage) actually forms a concave, laterally lipped roof over the cranial extremity of the stapes, while a distinct stapedial impression extends to a midforaminal level. The stapedial artery is of unusually small caliber; whether this reduction is an example of anatomic variation or of simple ontogenetic regression depends on the appearance of the vessel in subsequent stages of development.

The incudal extremity (the articular end of the future processus longus) comes almost in contact with the most caudal portion of the stapes; this process fuses solidly with the main incudal mass.

16 Mm. Embryo (Carnegie 5542 B).—The topographic features are little changed from those observed in the preceding specimen. The stapes maintains a blunt attachment to the hyoid bar, thicker and more irregular than in the 15.8 mm. stage (fig. 8); the stapedial artery is of relatively small diameter in its intercrural course. The approximation of the stapes and the incus (long process) is close.

From a reconstruction of a 16 mm. embryo, Jenkins [43] (1910) was able to describe the hyoid bar—still in a prechondral stage—as passing cranially on the inner side of the facial nerve; the cranial extremity of the hyoid bar had undergone bifurcation, the anterior portion being directly continuous with the stapes. The stapes at this stage was ring-like and consisted simply of concentrated mesoblast.

Broman [5b] (1899) observed the first evidence of separation of the malleus and the incus at this stage of development.

17 Mm. Embryo (Carnegie 5542 B).—In the 17 mm. embryo the otocyst is constricted to form two communicating cavities, an anterior, which is destined to be the membranous cochlea, and a posterior, lying opposite the stapes, which will be the membranous vestibule. Further transition of periotic mesenchyma into precartilage is evident, particularly on the medial and dorsal aspects of the capsule. The hyoid bar and Meckel's bar and the derivatives of the latter, namely, the malleus and the incus, show differentiation from precartilaginous into true cartilaginous tissue; the stapes, however, predecessor of both the capsule and the other ossicles, has only begun to show transitional histologic changes in the direction of precartilage. The stapes is annular in form, and the intercrural space appears quite round and regular.

The first definite approximation of the incus and the stapes occurs in this specimen.[42] At the level of the incudostapedial junction, the articular end of the incus appears to arise directly from the stapes itself, but under high power microscopic examination a line of demarcation can be distinguished between the two structures; an ill defined zone of radially arranged mesenchymal cells effects the separation. At a some-

43. Jenkins, G. T.: Some Features of the Auditory Apparatus of a 16 mm. Human Embryo as Shown in a Reconstruction Model, Proc. Roy. Soc. Med. (Sect. Otol.) **3**:34-35, 1910.

what lower level the body of the incus, now separated from Meckel's bar, and the long process are composed of young true cartilage; the distal lentiform portion consists of a young cartilaginous core with a peripheral cap of precartilage. This cap of precartilage is intimately applied to the transitional tissue of the stapes as mentioned hereinbefore; but there has never been any topographic evidence to warrant the supposition that the stapes effects a primary attachment to the long incudal process (Cleland,[44] 1889).

Only the most caudal extremity of the stapes is in contact with the hyoid bar. The small size of the stapedial artery is evidence that regressive change is taking place.

18 Mm. Embryo (Carnegie 409).—At this stage the stapes is definitely annular in form and its outline clear. The stapedial artery is relatively larger than in the preceding specimen but smaller than in certain of the early stages (10.6 mm., 13.1 mm.). The hyostapedial attachment has become narrower, so that a ligamentous character is suggested; however, as Broman [5b] stated, this connection ("interhyal") does not differentiate beyond the mesenchymal stage.

Somewhat below (estimated, 30 microns) the hyostapedial junction, the distal end of the incus (lenticular process) comes into close contact with the caudal extremity of the annulus stapedialis; under high power examination it is now impossible to distinguish between the cellular contributions of these adjacent structures. However, this apposition of incudal and stapedial elements involves only the peripheral dense mesenchymal envelope of each mass, the homogeneous zone thereby representing the primordium of the future articular capsule.

19.8 Mm. Embryo (Carnegie 1108).—The hyoid cartilage, appearing as an elongate structure, courses along the inferomedial aspect of the facial nerve, its distal end touching the lateral extremity of the stapes at right angles through the intermediation of a gradually constricting bridge of dense mesenchymal tissue, the hyostapedial "ligament." The long process of the incus courses dorsomedially to impinge on the lateral stapedial extremity immediately caudal to the hyoid attachment.

The malleus and the incus consist of young cartilage; the stapes is still in a stage of transition between dense mesenchyma and precartilage. The malleus and the incus are now distinctly separate "ossicles"; the deeply staining zone of tissue which forms the line of demarcation between them represents the future malleoincudal articulation capsule. Meckel's bar still retains a true attachment (anterior process) to the malleus.

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44. Cleland, J.: The Incus, Stapes and Styloid, in Cleland, J.; Mackay, J. Y., and Young, R. B.: Memoirs and Memoranda in Anatomy, London, Williams & Norgate, 1889, p. 173.

20 Mm. Embryo (Carnegie 462).—In the 20 mm. embryo the stapes is overhung cranially by a canopy-like projection of the lateral capsular wall, and this demonstrates again the developmental separateness of visceral and somatic skeletal elements. The canopy-like structure was first clearly observed in the 15.8 mm. stage and represents the lateral bulging of rapidly growing capsular tissue over the cranial aspect of the stapes, which in turn accentuates the process by becoming more intimately applied to the capsular wall. The stapes, still in an indefinite transitional stage but more nearly resembling precartilage than mesenchyma, is clearly distinguished from the surrounding capsular tissue by an intermediate zone of pale-staining, radially directed mesenchymal cells.

The long process of the incus, its distal end still consisting of precartilage, impinges on the lateral aspect of the stapes at the site of the future incudostapedial articulation (fig. 9). At a more caudal level (estimated, 80 microns) a wavy strand of deeply stained mesenchymal cells extends obliquely from the dorsolateral tip of the articular end of the stapes to the hyoid bar, lying ventrolateral to the facial nerve; this strand represents the greatly attenuated hyostapedial "ligament."

The portion of Meckel's bar which will become the malleus ascends superolaterally from the mandibular level to encroach on the inferomedial aspect of the body of the incus; a caudal projection of the malleus comes into the vicinity of the hyoid bar lateral to the facial nerve. Meckel's bar ascends only to the level of the caudal portion of the stapes. The hyoid bar, on the other hand, ascends from its proximal level to meet the caudal aspect of the posterolateral portion of the angular mass; it approximates the capsular cartilage, apparently fusing with it (the tympanohyal), then continues in an anterosuperior direction around the angle to meet the hyostapedial ligament. Both the mandibular and the hyoid bars are cartilaginous throughout.

22 Mm. Embryo (Carnegie 4960).—In the 22 mm. embryo the tissue of the stapes has changed from dense mesenchymal cells wholly into precartilage. The otic capsule on its lateral aspect consists predominantly of precartilage, particularly opposite the stapes and along the cochlear margin; the canalicular portion (angular mass) and the medial wall of the cochlear portion are cartilaginous. Rarefaction in the areas immediately surrounding the epithelial otic cavities is in the initial stage.

The cartilaginous long process of the incus approximates the lateral extremity of the stapes (fig. 10). At the same level a deeply stained structure extends from the incudostapedial region toward a triangular fragment of precartilaginous tissue lying lateral to the facial nerve—identifiable as the inferolateral extremity of the angular mass to which the hyoid bar becomes attached. After narrow attachment to the stapes

(hyostapedial ligament), the bar passes around the ventral aspect of the facial nerve to its medial side and then continues its caudal course.

The stapedial artery, quite as large as that of the 17 mm. embryo, maintains its identity as it passes through the obturator foramen but then becomes lost in the vascular mesenchyma along the lateral cochlear wall.

23.4 Mm. Embryo (Carnegie 4638).—The stapes is a distinctly precartilaginous annular structure; the intercrural space is round and regular and transmits a small stapedial artery, comparable in size and direction to the vessel in the preceding specimen. This description applies as well to the stapes of the 22.8 mm. embryo, studied by Anson, Karabin and Martin.[18a] The hyostapedial attachment persists as a dense band of deeply stained mesenchymal tissue extending between the lateral stapedial extremity and the junction of the hyoid bar with the angular mass; the "ligament" is the primordium of the stapedial tendon and muscle.

25 Mm. Embryo (Wisconsin 87).—The otic capsule at this stage is a distinct skeletal entity, even though manifesting a condition of histologic flux. The epithelial labyrinth is everywhere surrounded by precartilage which is undergoing a retrogressive change into mesenchymal syncytium preparatory to the formation of periotic spaces; the process is especially active in the region of the vestibule and the cochlea. In the angular mass the semicircular ducts are surrounded by concentric zones of reticulum and precartilage. The ventral portion of the cochlea is likewise precartilaginous; this tissue extends from the lamina basalis laterally to a point opposite the facial nerve, including the area immediately adjacent to the stapes. Young true cartilage constitutes the entire dorsal (neural) portion of the cochlear capsule and the canalicular area, with the exception already mentioned, of zones of precartilage about the epithelial canalicular ducts.

The squamous portion of the occipital cartilage (lamina parietalis) attaches directly to the dorsolateral capsular angle; the basilar portion (lamina basalis) joins the capsule at the dorsomedial extremity.

The stapes itself, entirely precartilaginous, lies partly embedded in the ventral precartilaginous mass of the capsule, just medial to the facial nerve; the extracapsular portion of the stapes is enveloped by the loose, vascular mesenchyma filling the future tympanic space. Concomitant growth of the stapes and the capsule has caused the cranial portion of the "ossicle" to be pressed more firmly into the lateral capsular wall, so that at this stage only a thin condensation of mesenchymal and precartilaginous cells about the periphery of the enclosed stapes permits that structure to be distinguished from the precartilage tissue of the adjacent capsule (fig. 11; arrow indicates width of primordial lamina stapedialis).

The stapes has a roughly annular form, with poorly defined capital, crural and basal elements. It demonstrates even at this early stage an oblique relationship to the otic capsule in which it is embodied; both its dorsoventral and its mediolateral axis are directed toward the middle line of the head. The capital extremity, identifiable as such only because of its relation to the incudal cartilage, is pear shaped; the rounded medial half is formed by the relatively massive medial (later, anterior) crus, the tapering lateral half, by the slender lateral (later, posterior) crus. The base is a bluntly oval mass, entirely embedded in the stapedial impression of the capsule. This orientation permits certain simple measurements: The distance from head to base is 0.392 mm.; the basal length, 0.32 mm.; the circumference of the medial (anterior) crus, 0.456 mm., and that of the lateral (posterior) crus, 0.632 mm. The crural lengths cannot be determined with accuracy because of the indefiniteness of the capital and basal junctions at this stage.

This typical precartilaginous stapes is characterized by rather densely packed cells whose fibrillar ramifications are only vaguely discernible in a homogeneous intercellular material; the darker appearance (as compared with cartilage) is due not to increased affinity for the stain but to more compact arrangement of nuclei. Compared with the reconstructed stapes of the 22.8 mm. stage (fig. 1, Anson, Karabin and Martin [1a]), the stapes of the 25 mm. embryo shows an increase in height of 0.098 mm. and an increase in basal length of 0.072 mm. The ratio of the head to base length to the basal length in the two specimens follows an accurate mathematical proportion. The intercrural or obturator space is a tiny oval hiatus with its long axis directed in the plane of the crura; it affords passage for a rudimentary stapedial artery. After traversing the foramen, the artery courses posterior to the facial nerve, i. e., between the nerve and the groove for this nerve, and immediately loses its identity in the spaces of the tympanic mesenchyma.

The vestibular (oval) window is present only as a roughly oval indentation on the lateral aspect of the otic capsule opposite the site of the future vestibular cistern; its outline must closely follow the contour of the enveloped portion of the stapes because of the intimate relationship of the structures involved.

Both the incus and the malleus are composed of young cartilage consisting of loosely arranged cells; the cytoplasm is pale staining, and the nuclei are large. The incus is structurally independent of Meckel's bar, and the future malleoincudal articulation is well defined. The incudal mass (body of the incus) is closely applied to the flattened ventrolateral capsular angle (future mastoid region), while its tapering medial end (processus longus) approaches the inferolateral extremity of the stapes; the future articular surface is represented by a well defined shallow depression on that portion of the stapes.

The first visceral pouch, representing the primitive auditory tube, is a slender elongate evagination of the dorsolateral pharyngeal wall, extending laterally into the tympanic mesenchyma to a point almost opposite the stapes (fig. 11).

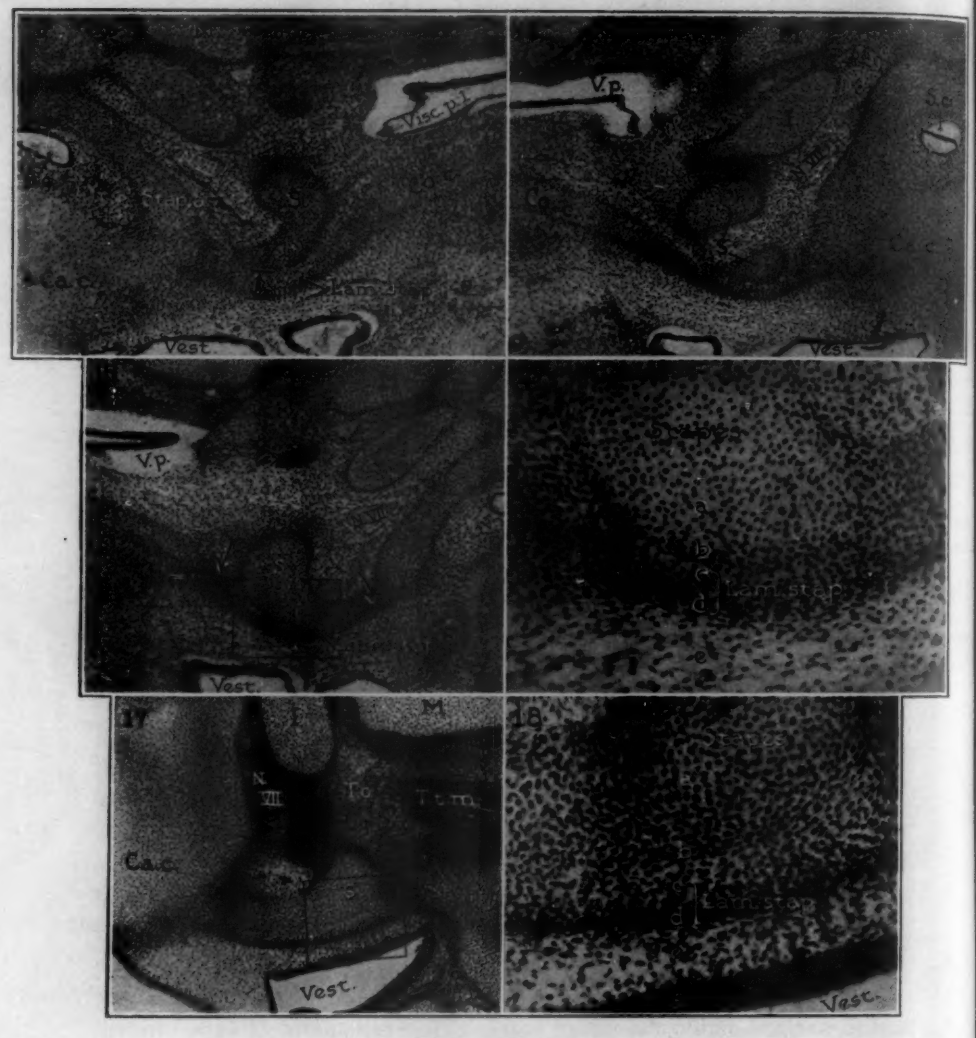
27.5 Mm. Fetus (Carnegie 2561).—In the 27.5 mm. fetus a second transitional phase in the histogenesis of the stapes is in progress. From the 7.0 mm. stage of the embryo, in which the stapes makes its first appearance, to the 18.0 mm. and 19.8 mm. stages an ill defined aggregate of mesenchymal cells was gradually molded into a denser unit of annular form without loss of the essentially mesenchymal character of the constituent tissue. Differentiation into precartilage has been observed during the 18.0 to 25.0 mm. stages. A widened but irregular strip of mesenchymal tissue extends from the posterior aspect of the capital extremity of the stapes to the approximate point of junction of the hyoid bar and the angle of the canalicular portion of the capsule. The histologic transition from precartilage to cartilage observed in this stage is in accord with Streeter's [45] account of the change in the otic capsule; there is gradual increase in the quantity of matrix combined with more definite encapsulation of nuclei, so that the new tissue more closely resembles true cartilage than precartilage (fig. 12). The matrix of young true cartilage stains poorly in contrast with the chromophilic character of the more mature type (figs. 17 and 19).

The stapedial artery may still be identified as a small vessel traversing the obturator foramen and terminating at right angles in an artery (stylomastoid) which courses along the caudal aspect of the facial nerve.

30 Mm. Fetus (Wisconsin 92).—While the otic capsule has increased in over-all dimensions, its shape is essentially that of the capsule in both the 25 mm. and 27.5 mm. stages. Precartilage has been replaced by true cartilage in the capsular wall except for two regions: (1) the lateral third of the anterior cochlear wall, consisting of young cartilage extending lateralward to blend into the precartilage of the future fenestral margin, and (2) the deeply stained compact layer (lamina stapedialis) which separates the stapes from the vestibule. Further dedifferentiation of periotic precartilage is evidenced by the widening of periotic channels and the accumulation therein of rather dense reticulum. In the interval between the lamina stapedialis and the membranous saccule, however, there is marked vacuolation of the syncytium with formation of numerous scattered spaces. As Streeter [45] pointed out, these multiple disconnected hiatuses are precursors of the periotic vestibular cistern, which is the first space to be established.

45. Streeter, G. L.: The Histogenesis and Growth of the Otic Capsule and Its Contained Periotic Tissue—Spaces in the Human Embryo, Contrib. Embryol. (no. 20) **7**:5-54, 1918.

Further critical features of stapedial development are demonstrated in this stage. The transition from the 25 mm. through the 27.5 mm. to the 30 mm. stage is marked by increase in intercellular matrix and by



Figures 13 to 18

(See legend on opposite page)

more discrete encapsulation of the cartilage cells, the change permitting definite tinctorial and histologic distinctions. The stapes is now a circumscribed annular mass of young true cartilage bearing a closer cellular resemblance to that of the adjacent cochlear wall than to that of the

more mature canalicular or incudal cartilage. Topographically considered, the general shape of the stapes in the 25 mm. embryo has been preserved, with some modifications. From an examination of sections (figs. 13, 14 and 15) through the midcrura and the midforamen certain conclusions can be drawn: The medial crus is wider and therefore greater in circumference than the lateral crus; the capital extremity is somewhat more massive than the base; the obturator foramen has increased markedly in size over that of the 25 mm. stage. One of the most interesting features of this stage is the development of the lamina stapedialis. In the 25 mm. embryo a bridge of precartilage extends between the cochlear and the canalicular portion of the capsule and separates the stapes from the labyrinthine otic cavity (fig. 11); in the 30 mm. fetus this bridge, thinner and more deeply stained, gives the appearance (in sections) of being slung hammock fashion between the precartilaginous ends of the opposing cochlear and canalicular walls (future fenestral margins; fig. 14). The base of the stapes is intimately applied to the surface of this lamina (fig. 15) but can be readily distinguished from it because of the concentrically arranged layer of cells on the basal periphery—a primitive perichondrium. Under high power examination (fig. 16) the lamina appears to consist of two cellular zones or stratums: (1) a pale-staining layer in apposition to the basal perichondrium of the stapes (*c* in fig. 16) and (2) a dense layer of fibroblastic tissue stretched completely across the future fenestra vestibuli (*d* in fig. 16). Within the first of these layers an intercellular reticulum can be identified; the nuclei are comparable to those of the embryonic mesenchyma in either the vestibular or the tympanic region but especially

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EXPLANATION OF FIGURES 13 TO 18

Transverse sections through the stapes and the adjacent capsular region in fetuses of 30 mm. to 34 mm. (figs. 13 to 16, 30 mm.; figs. 17 and 18, 34 mm.); figs. 13 to 15 and 17, × 17; figs. 16 and 18, × 65. Figure 13 shows the stapes and the lamina stapedialis (interval between tips of arrow). Figure 14 shows the annular stapes close to the long process of the incus; there is dedifferentiation of capsular tissue at the sites of the labyrinthine spaces. Figure 15 shows the stapes impinging on the lamina stapedialis (limits of lamina marked by arrows). Figure 16 presents the area demarcated in figure 15 at high magnification. At *a* one may see young true cartilage of the stapes; at *b*, the limiting zone of fibroblasts; at *c*, the outer layer of the lamina; at *d*, the inner layer of the lamina (primordium of the vestibular perichondrium); at *e*, loose mesenchyma in the vestibular cavity. Figure 17 shows the anterior one third of the stapes. Figure 18 presents the area demarcated in figure 17 at high magnification. At *a* one may note stapedial cartilage; at *b*, the site of the primordial basal perichondrium; at *c*, the outer zone of the stapedial lamina; at *d*, the inner zone of the lamina; at *e*, the loose reticular mesenchyma of the vestibule; at *f*, the epithelial lining of the vestibule (primordial saccule).

to those in the latter, with which the layer appears to be in continuity around the edges of the stapedial base. That these cells are of a primitive character and derived from the outer surface of the original precartilaginous bridge and that they simply occupy the interval between two actively growing (or differentiating) structures—the stapes and the lamina stapedialis—seems to be their likely interpretation. The second layer, of fibroblastic tissue, blends evanescently with the vestibular periotic reticulum on its inner surface and with the pale-staining layer of primitive perichondrium on its outer surface, and has undoubtedly been formed by dedifferentiation and active growth of the original precartilage bridge hitherto mentioned.

The stapedial artery can still be traced through the obturator foramen, but its exceedingly small size indicates that it is in a terminal involutionary stage.

Meckel's cartilage maintains a dense connective tissue attachment to the head of the malleus. The main incudal cartilage mass (body of incus) is closely applied to the anterolateral capsular angle; its processus brevis projects inferiorly to make contact with the capsule; its processus longus extends medially to touch the capital extremity of the stapes, but no true articulation as yet exists between the approximated surfaces.

The auditory tube has increased in caliber and extent; laterally its bifid extremity partially envelops the manubrium of the malleus.

In the 30.7 mm. fetus (Carnegie 4750) the general features of the otic capsule and the stapes are similar to those of the corresponding portions of the developing auditory apparatus in the 30 mm. specimen. The stapedial muscle, though still rather indefinite at its proximal end, passes toward the capsule and is a relatively well developed structure as it courses along the medial aspect of the facial nerve. The tendinous attachment of the muscle to the posterior surface of the capital extremity of the stapes is more definite than is its fixation to the capsular wall.

32 Mm. Fetus (Carnegie 5154).—The stapes is still a discrete cartilaginous ring invested by a peripheral condensation of mesenchymal cells forming a primitive perichondrium. As was seen in the 30 mm. series (fig. 15), the "perichondrial" investment of the stapedial base—that portion in intimate contact with the lamina stapedialis—formed a demarcation line (*b* in fig. 16) between the cartilaginous base (*a*) and the outer pale-staining layer of the lamina (*c*). A similar zonal arrangement exists in the 32 mm. stage as well.

34 Mm. Fetus (Carnegie 6647).—The stapes is no longer of simple annular form; it has been modified to the extent of exhibiting a flattened nodular capital extremity and a substantially widened base. The first indication of the change from a ring-shaped to a stirrup-shaped structure is detectable in the 34 mm. stage (compare the 40 mm. fetus, figs. 3

and 6, Anson, Karabin and Martin[18a]). In the stapes of the current stage (fig. 17) a further step in metamorphosis has occurred. The simple uninterrupted growth in the annulus stapedialis with further differentiation of its perichondrial investment and distinct separation from the adjacent lamina is now accompanied by alteration within the lamina. The basal perichondrium (the narrow basal layer of radially directed, deeply stained mesenchymal cells) observed in the 30 mm. and 32 mm. fetuses has become virtually nonexistent, with the result that the bare cartilaginous base is left in juxtaposition with a pale-staining layer of cells previously represented as the superficial stratum of the lamina stapedialis (fig. 16, *b;* fig. 18, *c*). The perichondrium-free base is in the process of active growth, nevertheless, as evidenced by thickening concentric layers of young cartilage on its surface. The pale-staining layer with which the growing base comes in contact has the histologic appearance of transitional tissue, since it is a mixture of young true cartilage, precartilage and primitive mesenchymal cells.

This is much in keeping with the observations made by Schmid[46] (1928), who stated that the stapes in

> . . . the earliest stage appeared as a ring and was clearly differentiated from the otic capsule. The zone of approximation between the stapes and the capsule is distinct in all stages and in all planes of section; the outline of the ring is quite sharp; the ring stains deep blue with hemalum-eosin, and the capsule appears pink. There is a histologic differentiation in the corresponding stages of development of the ring and the capsule.

He expressed the opinion that concomitant stapedial and capsular growth results in the apposition of these structures and that observations on tinctorial differentiations militate against the assumption that the stapes arises wholly from the capsule (Gruber, Fuchs and others). However, he assumed that no perichondrial investment of the stapedial base ever existed, on the ground that the rapid growth of capsule and stapes would prevent this differentiation as the two parts became approximated. It must be pointed out that the earliest stage studied by Schmid was the 37 mm. one; the present authors have demonstrated that in the 34 mm. fetus the primitive perichondrium of the base has already undergone deorganization, thereby permitting the cartilaginous stapedial base to fuse with the outer cellular layer of the lamina stapedialis.

At the angle of junction of the vestibule with the scala vestibuli there is a narrow wedge-shaped area of persistent dense mesenchyma where chondrification is delayed; it projects anteriorly into the cochlear cartilage for a short distance (estimated thickness, 80 microns). This

46. Schmid, H.: Beitrag zur Entwickelungsgeschichte des Stapes, Anat. Anz. **66**:109-115, 1928.

is the future site of the fissula ante fenestram—described by Bast[47] (1933) in the 43 mm. fetus.

43 Mm. Fetus (Wisconsin 90).—The otic capsule of the 43 mm. fetus consists chiefly of mature cartilage. Regions of delayed chondrification occur only around the semicircular canals, in the interval between the fenestral margin and the stapedial base and at the angle of junction of the vestibule with the scala vestibuli. The persistence of precartilage about the canals is associated with the growth of these channels and with the formation of periotic spaces around the membranous ducts (Streeter[46]; Bast[47]; Foley[48]). The vestibular periotic cistern is now discernible, although occupied by loose-meshed reticulum; the cochlear periotic spaces are not as well defined.

Since the stapes and the lamina stapedialis are so intimately related in their developmental history, they will be described together. The capital extremity of the stapes has been further modified to form a distinctly flattened articular surface, short columnar crura and an elongate flattened base. The lamina stapedialis has become markedly thinned, as a result, no doubt, of the rapid expansion of the stapedial base. The lamina consists of an inner compact layer of fibroblastic tissue, representing the primitive perichondrial investment of the vestibule, and of an outer layer consisting of transitional cells. The inner compact layer is in contact with the few mesenchymal cells remaining in the newly formed periotic vestibular cistern. The lamina no longer has the appearance of being slung from the margin of the window but is immediately continuous with the perichondrial investment of the cochlear and canalicular portions of the vestibule. In the same way, the perichondrium covering the tympanic portion of the stapes, since it no longer invests the base, becomes continuous with the perichondrial layer over the outer labyrinthine wall adjacent to the fenestra vestibuli. In form and structure the stapes of the 39.5 mm. (Carnegie 6203) and of the 40 mm. fetus (Anson, Karabin, and Martin[18]) closely resembles that of the 43 mm. fetus herein discussed.

The fissula ante fenestram was identified for the first time in the preceding (34 mm.) specimen. The fissula in the present series of sections is represented by a prominent vestibular portion packed with fibroblastic tissue which is continous with the vestibular perichondrium (fig. 19). This narrow channel extends into the otic capsule and consists of modified precartilage surrounded by pale-staining young true

47. Bast, T. H.: Development of the Otic Capsule: II. The Origin, Development and Significance of the Fissula Ante Fenestram and Its Relation to Otosclerotic Foci, Arch. Otolaryng. **18**:1-20 (July) 1933.

48. Foley, J. O.: The Cytological Process Involved in the Formation of the Scalae of the Internal Ear, Anat. Rec. **49**:1-14, 1931.

cartilage. No connection is yet established with the tympanic cavity; the fissular tissue simply fades away as the sections pass cranially.[49]

While areas of delayed chondrification still appear at this stage, there is at the same time evidence of degenerative change in the midst of the canalicular cartilage mass. In the 25 mm. embryo the angular mass consisted of young cartilage, while the remainder of the capsule was in a more primitive histologic state. In the subsequent stages the cartilage cells in this area have taken on a loosely knit, pale-staining appearance. And now, in the 43 mm. stage, the oldest cartilage tissue in the otic capsule anticipates the ultimate differentiation into bone by undergoing swelling and patchy avascular necrosis of its constituent cells.

50 Mm. Fetus (Wisconsin 86).—The cartilage comprising the otic capsule is almost wholly of the mature type; the exceptions are again the pale-staining cartilage of the fenestral margins and the wedge-shaped area of invaginated periotic tissue which is the primordial fissula ante fenestram.

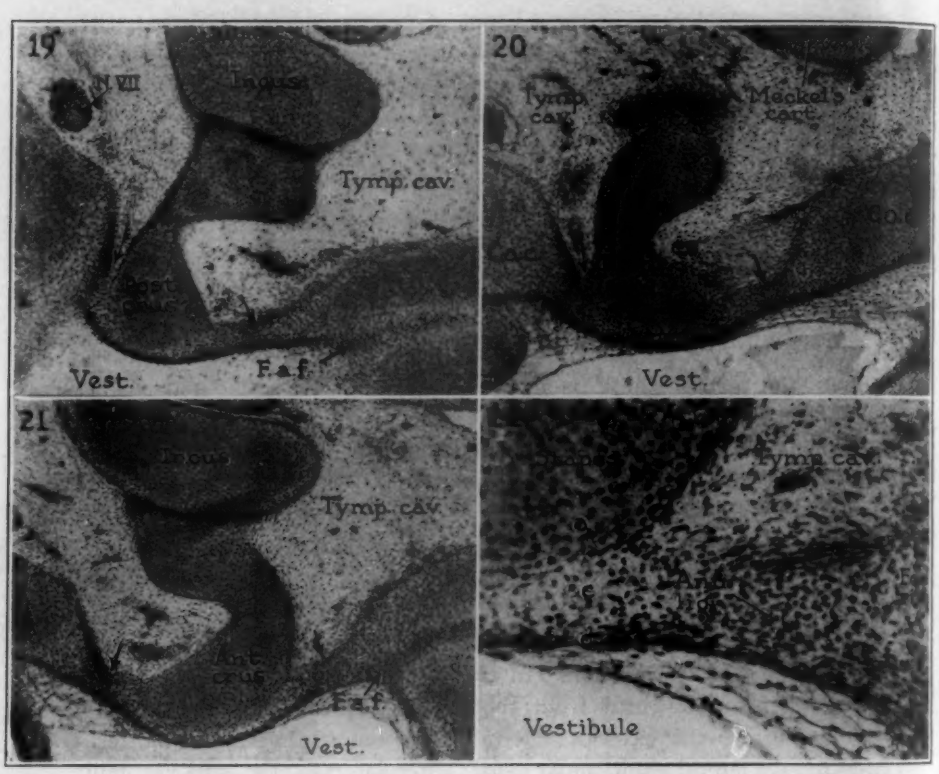
The periotic vestibular cistern is capacious; the membranous otic cavities—the saccule and the utricle—occupy only a fraction of the available space. Loose-meshed reticulum persists to some extent over the walls of the vestibule and extends anteriorly into the scalae and posteriorly into the semicircular canals. The embryonic mesenchyma occupying these channels is markedly vacuolated, however, and the formation of definite periotic spaces appears imminent.

As determined by reconstruction, certain morphologic features which were altogether lacking in the 25 mm. stage are now present. In the cochlear portion of the capsule the spiral septum is well developed and separates the first turn of the cochlea from the second; the upper aspect of the promontory projects beneath the stapes and approaches the inferior fenestral margin. In the vestibular portion of the capsule, six distinct orifices lead from the vestibule into other parts of the labyrinth or to the exterior; laterally, the fenestra vestibuli; posterolaterally, the lateral semicircular canal; anterolaterally, the scala vestibuli;

49. An excellent historical review of the literature pertaining to the fissula ante fenestram is given by T. H. Bast (Ossification of the Otic Capsule in Human Fetuses, Contrib. Embryol. [no. 121] **21**:52-83, 1930). Careful studies on the origin and development of this structure have been made by Bast (1933[47]; Development of the Otic Capsule: III. Fetal and Infantile Changes in the Fissular Region and Their Probable Relationship to the Formation of Otosclerotic Foci, Arch. Otolaryng. **23**:509-525 [May] 1936), B. J. Anson and J. Martin (Fissula Ante Fenestram: Its Form and Contents in Early Life, ibid. **21**:303-323 [March] 1935), J. G. Wilson (Fissula Ante Fenestram and the Adjacent Tissue in the Human Otic Capsule, Acta oto-laryng. **22**:382-392, 1935) and B. J. Anson and J. G. Wilson (Structure of Petrous Portion of Temporal Bone, with Special Reference to Tissues in Fissular Region, Arch. Otolaryng. **30**:922-942 [Dec.] 1939).

anteromedially, the superior opening of the medial acoustic meatus (the future superior cribriform area); posteromedially, the aquaeductus vestibuli and immediately lateral to the aquaeductus, the superior semicircular canal. The two latter channels are separated by a thin perpendicular plate of cartilage.

The stapes has increased only slightly in size over that of the 43 mm. stage. Its topographic features, however, are better developed. It possesses a short but well defined capital extremity, which demonstrates



EXPLANATION OF FIGURES 19 TO 22

Transverse sections through the stapedial and fissular regions in fetuses 43 mm. and 50 mm. in length (fig. 19, 43 mm.; figs. 20 to 22, 50 mm.); figs. 19 to 21, × 21; fig. 22, × 75. Figure 19 shows secondary fusion of the stapes and the otic capsule and incorporation of the lamina into the stapedial base. The fissula ante fenestram is an ill defined zone of delayed chondrification. Figures 20 and 21 show the stapes, now with a distinct head, crura and a base. The lamina stapedialis has been incorporated into the base; arrows indicate the limits of the annular ligament. Figure 22 shows a portion of the stapedial base and annular ligament at high magnification. The zone of transitional tissue between the stapes and the fenestral rim is the primordium of the annular ligament; at *a* one may see mature cartilage of the stapedial base; at *c*, young cartilage of the base, at *d*, perichondrium of the base, continuous with the annular ligament and the vestibular perichondrium; layer *b*, seen in figure 10, has been obliterated.

for the first time a shallow foveate articular surface (figs. 20 and 21). The crura are slightly bowed; the anterior is somewhat shorter and less massive than the posterior crus. The obturator foramen has a definitely oval form, its long axis directed in the plane of the crura. It is occupied by densely vascularized mesenchyma, but there is no stapedial artery. The base of the stapes has increased greatly in size since the 25 mm. stage. It is now somewhat flattened on its vestibular side and narrowed and gently rounded on the tympanic aspect. It is roughly oval, the anterior being slightly wider than the posterior portion. The first suggestion of a stapedial rim makes its appearance in this specimen. The rim can be identified as a low narrow ridge on the superior basal margin and as a rather prominent posterior projection at the junction of the posterior crus with the base. There is only the slightest indication of a rim or bulge on the inferior basal margin and at the anterior extremity of the stapes. In histologic appearance the stapes is similar to that of the preceding stage except for the tissue of the base. In the 43 mm. fetus a pale-staining zone of transitional cell character was identifiable in the stripe between the perichondrium-free stapedial base and the deeper fibroblastic layer. In the 50 mm. fetus that layer has been virtually obliterated and its place taken by a pale-staining layer of young true cartilage—thin at the center, thicker at the periphery of the base. Seemingly, with the deorganization [50] of the original perichondrium of the stapedial base there followed a period of interstitial basal growth, with resultant encroachment on the receding lamina. Whether the aforementioned transitional zone has chondrogenous potentialities and is added to the base of the stapes or is gradually compressed to be eventually obliterated by the advancing base cannot be definitely decided until closely graded intermediate stages are available for study. Nevertheless, since the base of the stapes steadily approaches the deep laminar remnant, it may be concluded provisionally at least that the lamina becomes applied to the cartilaginous base and represents its secondary but true perichondrial investment. In this way the perichondrium covering the base of the stapes becomes perfectly continuous with the layer of dense mesenchymal (fibroblastic) tissue which extends between the base and the fenestral margin, and with the perichondrium of the vestibule; similarly, the primitive perichondrium investing the capital and crural portions of the stapes is continuous not with the base but with

50. Dr. L. B. Arey has suggested the term "deorganization" rather than "dedifferentiation" to describe the recession of the primitive perichondrial investment of the stapedial base (30 mm. stage); the cells simply change from an organized compact layer to a diffuse layer of primitive mesenchyma (34 mm. and 43 mm. stages). This change in concentration of tissue is not the cellular transition that occurs in the dedifferentiation of a specialized into a less specialized tissue, i. e., cartilage into precartilage and precartilage into embryonic mesenchyma.

the dense mesenchymal tissue between the base and the fenestral margin and with the perichondrium of the adjacent tympanic surface of the labyrinth (fig. 21). As a result of these new perichondrial relationships, the base of the stapes is separated from the fenestral margin only by the altered precartilage of the original lamina stapedialis—at the site of the future annular ligament (fig. 22); the stapes during this stage of development (34 mm. to 50 mm.) is therefore histologically continuous with the fenestral margin of the otic capsule.

Concerning the development of the annular ligament, since a hiatus now exists in available material between the 50 mm. and the 75 mm. stage, only possibilities can be considered. If it is assumed that the precartilage of the original lamina does play a role in the formation of the opposing stapedial and fenestral rims, it is conceivable that with gradual recession and condensation as a result of concomitant stapedial and fenestral growth this tissue may undergo dedifferentiation into mesenchyme (fibroblastic tissue) and eventually assume a ligamentous character; or, with gradual depletion of the precartilage, tissue may be recruited from the adjacent tympanic and vestibular perichondrial layers to form an annular ligament passing through the regular stage of radial arrangement.

The vestibular window no longer has a triangular configuration (compare 40 mm. stage, fig. 2, Anson, Karabin and Martin [18]); it is roughly oval, conforming closely to the margin of the stapedial base. The superior and inferior fenestral margins are sharp; the anterior and posterior extremities are blunt and continuous with the corresponding capsular walls.

The fissula ante fenestrum closely resembles that of the 43 mm. stage. Its vestibular orifice [51] is prominent and is filled with fibroblastic tissue (fig. 21). The fissular portion extends into the capsular cartilage for a maximum distance of 0.4 mm. but fails to communicate with the tympanic cavity. Fissular connective tissue courses upward through twenty consecutive sections, from the vestibular extremity to the termination near the tympanic surface of the promontory (distance, 0.3 mm.).[52]

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51. Here, of course, the term "orifice" is employed—as it commonly is in anatomy—for convenience of description. The name here is applied to an extremity of a fault in the cartilage of the capsule. Obviously there is neither a true fissula nor a true orifice of the latter unless all contained fibrous tissue is first removed to leave a channel within the cartilaginous otic capsule. Comparably, there would be no fissula in the adult osseous capsule until the temporal bone was boiled. Actually, the fissula was first observed in this way, the investigator passing a bristle into its minute opening as one might explore a cranial foramen or a vestibular window in a specimen of "cleaned" skull.

52. Bast [47] summarized the formation of the varied fissular tissues as follows: "The tissue of the fissula arises, as does the periotic tissue, from precartilage.

(Footnote continued on next page)

SUMMARY

The cytoblastema of the stapes makes its first appearance in man at the 7 mm. stage. It arises from the cranial extremity of the second visceral (hyoid) bar as a bulbous mesenchymal mass; in this earliest primordial state it is not perforated by an artery. The stapedial blastema is located lateral, as well as slightly caudal, to the otocyst; it is wholly separate from the epithelial vesicle (otocyst) and from the meager condensation of mesenchymal cells about the latter's periphery. At the 8 mm. stage a single small vessel perforates the cytoblastema, but a ring-shaped form is not attained until the 12 mm. or 13 mm. stage is reached. Then rather rapid absorption of periarterial mesenchymal tissue results in the formation of a distinct obturator foramen in the 13.95 mm. embryo. From the initial appearance of the stapedial blastema until a condition is attained in which a distinct forerunner of a stapedial tendon and muscle becomes apparent, a connection between the stapes and the hyoid bar (or a modification of it, i. e., the pyramidal eminence) is maintained; the hyostapedial attachment, however, fails to differentiate histologically beyond the stage of dense mesenchymal tissue. In the 18 mm. and 19 mm. stages the differentiation of the dense mesenchymal tissue of the annulus into precartilage is under way; the transformation is complete in the stapes of the 22 mm. embryo.

The second developmental step in stapedial histogenesis is taken in the 27.5 mm. embryo—precartilage then undergoes further differentiation into true cartilage. In the 30 mm. fetus the young true cartilage has wholly replaced the precartilage.

At 34 mm., but more clearly at 43 mm., the distinctive portions of the definitive stapes are discernible; a somewhat flattened medial part, the future base, is set off from adjoining bowed portions, the crura; a stumplike capital extremity is present, but a neck as a definite division is not yet identifiable.

In the 50 mm. fetus, the stapes has become roughly stirrup shaped; distinct crura, a base and a short but well molded head are present; a very shallow foveate articular surface makes its appearance.

Approximation of the blastema of the stapes to that of the otic capsule is a secondary feature of the concomitant growth of both structures. The stapes approaches the periotic tissue at the 10.6 mm. stage and soon (in the 12 mm. stage) is in intimate contact with it. In the 15.8 mm. stage the medial extremity of the stapes impinges on the lateral capsular wall so that a thick roof, concave on the under side,

1. The precartilage undergoes a regressive change to form a vacuolated reticular tissue. 2. With the entrance of blood vessels from the middle ear, the tissue undergoes new growth, filling the fissula with fibroblasts which form a fine fibrillar connective tissue. 3. At the periphery of the fissula this connective tissue changes to cartilage, whereas in the center of the fissula a loose vacuolated vascular connective tissue is formed."

is formed over the stapes and a distinct impression produced to a midforaminal level. The portion of the stapedial cartilage in contact with the capsule is destined to be the basal portion of the future ossicle. The basal extremity in the 25 mm., 27.5 mm. and 30 mm. stages is closely applied to the anterolateral labyrinthine wall, separated from it by a thin peripheral condensation of mesenchymal cells—the primitive perichondrium. In the 34 mm. fetus the primordial perichondrium of the base has been obliterated, leaving the cartilaginous base in juxtaposition with the lamina stapedialis. Active growth in the region of the base is evidenced from this stage onward. In the 43 mm. and 50 mm. fetuses vigorous interstitial growth brings the base into even closer apposition with the dense fibroblastic remnant of the lamina. This layer is eventually applied to the vestibular surface of the base as a secondary but true perichondrial layer. It is apparent that the stapedial rim or flange (and the fenestral rim), which appears for the first time in the 50 mm. specimen, is derived chiefly from the precartilaginous remnant of the lamina stapedialis located between the base and the fenestral margin. In this way a portion of the stapedial base and the perichondrial investment of the vestibular surface of the base are derived from the original capsular tissue.

The crura become definable when the capital and basal extremities attain a protuberant and a flattened form, respectively. They are first identifiable in the 34 mm. stage. Characteristic bowing and shortening of the anterior crus are definitely established in the 50 mm. fetus. Although a capital extremity is first represented in the 34 mm. fetus, a definite head, appearing as a flattened nodule set off only moderately well from the crura, is to be found somewhat later, in the 43 mm. specimen. A foveate articular surface appears in the 50 mm. stage.

The annular ligament with the exception of the fissula ante fenestram is the most inconstant structure encountered. Its site in the 43 mm. and 50 mm. stages is represented by the peripheral remnant of the lamina stapedialis. We could not definitely ascertain whether its definitive form is attained through dedifferentiation and condensation of laminar precartilage or through invasion of fibroblasts from the adjoining perichondrial layers; possibly both processes are involved.

A vestibular (oval) window cannot be demonstrated in the earlier stages because of the intimate relationship of the stapedial cytoblastema with the surrounding capsular tissue; it would be represented merely by an oval depression in the anterolateral labyrinthine wall. The development of a lamina stapedialis in the 25 mm. and 30 mm. stages prepares for the appearance of a future fenestra but does not represent the window itself. A true fenestra vestibuli occurs in the 43 mm. fetus.

The fissula ante fenestram is histologically a region of delayed chondrification formed by a cleftlike invagination of periotic tissue into

the capsular cartilage at the junction of the vestibule and the scala vestibuli. Its first appearance is made in the 34 mm. fetus. In primordial form it is a wedge-shaped mass of altered precartilage; the vestibular orifice is prominent and packed with fibroblastic tissue derived from the adjacent vestibular perichondrium. A tympanic orifice was not observed in any of the later stages studied (34 mm., 43 mm., 50 mm.).

To conclude: The definitive human stapes is derived from two sources: the second visceral (hyoid) bar and the lateral capsular wall. The origin in the visceral bar is primary and provides the greater mass of the stapes—the annulus stapedialis; subsequent fusion with the lamina stapedialis of the otic capsule and secondary differentiation of laminar tissue provide the stapedial rim, the vestibular surface of the base and the basal perichondrium. Further modification of the peripheral laminar tissue leads to the formation of the annular ligament of the stapes.

RELATION OF THE EUSTACHIAN TUBE TO CHRONIC PROGRESSIVE DEAFNESS

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It is an established fact that blockage of the eustachian tube plays an important role in the development of chronic progressive deafness. Coates [1] stated that most of the chronic progressive deafness which becomes evident in adult life has an origin in a neglected eustachian tube or inflammation of the middle ear in childhood. Crowe and Baylor [2] stated that a long-continued partial obstruction of the eustachian tube in children causes retraction of the tympanic membrane, impaired hearing for high tones, with relatively good hearing for low tones, and sometimes total loss of hearing by bone conduction.

The eustachian tube was treated for this type of deafness as far back as 1654 by Tulpus, who first described blockage of the eustachian tube in relation to certain types of deafness. Valsalva in 1707 described self inflation of the eustachian tube to act as a means of clearing mucus from the ear and as an aid in the treatment of deafness. Jonathan Wathen in 1755 catheterized the eustachian tube and elaborated on this procedure in relation to deafness. Mayer, of Copenhagen, in 1868 first advised the removal of adenoids toward relief of otitis media. Politzer in 1898 introduced the air douche for inflammations of the eustachian tube and middle ear. Killian later developed intranasal surgical technic and stressed the importance of a healthy nose in relation to maintenance of normal hearing. Henle in 1866 first accurately described the microscopic anatomy of the tube and illustrated its position in the bony skull and the relationship of the muscles to the tube. During the eighteen seventies many observers discussed the physiology of the tube, as to whether it was open or closed. It was later proved by Rich,[3] in 1920, that the tube normally is closed and that it opens during swallowing, yawning and sneezing reflexes.

Presented as a candidate's thesis to the American Laryngological, Rhinological and Otological Society, Inc., Oct. 30, 1941.

1. Coates, G. M., in Jackson, C., and Coates, G. M.: The Nose, Throat and Ear and Their Diseases, Philadelphia, W. B. Saunders Company, 1929.

2. Crowe, S. J., and Baylor, J. W.: Prevention of Deafness, J. A. M. A. **112** 585 (Feb. 18) 1939.

3. Rich, A. R: Bull. Johns Hopkins Hosp. **31**:206 (June) 1920.

The anatomic position of the pharyngeal orifice predisposes the eustachian tube to infections and involvement by conditions of the nose, sinuses and lower part of the respiratory tract. The nasopharynx is essentially a part of the nose and is lined by the same ciliated mucous membrane. It conveys air and adds to the local defense measures, acts as barrier between the nose and the pharynx proper by its musculature, drains secretions from the ear cavities and receives drainage from the nose.

The chief function of the eustachian tube is to equalize the air pressure of the tympanic cavity with that of the outside atmospheric

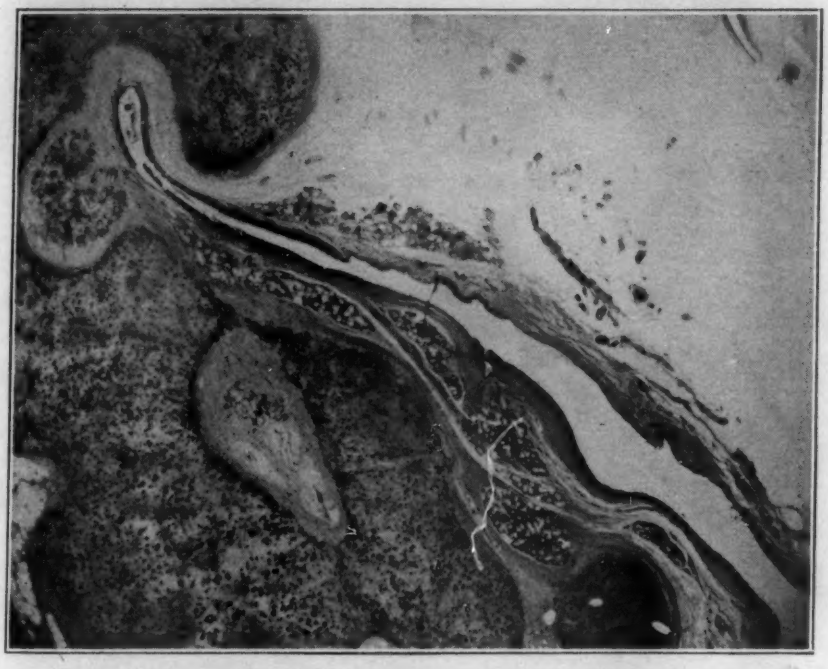

Fig. 1.—Cross section of a normal eustachian tube.

pressure. It serves, moreover, to drain secretions from the middle ear cavities. This is accomplished by the ciliary activity of the mucosa. The direction of the motion of the cilia is toward the pharyngeal opening. The tube also serves to protect the middle ear from foreign bodies and inflammations from the nasopharynx.

The eustachian tube is composed of two parts, one bony portion and one cartilaginous portion, and is about 36 mm. in length; its direction is downward, forward and medialward, and it forms an angle of about 45 degrees with the sagittal plane. The bony portion, about one-third

its length, begins at the carotid wall of the tympanic cavity and extends about 12 mm., gradually narrowing, ending at the angle of the junction of the squamosa and petrous portions of the temporal bone. The cartilaginous portion comprises about two-thirds its length, or 24 mm. It extends from the junction of the osseous portion on to the pharyngeal orifice, which is located along the lateral pharyngeal wall under the mucous membrane, where it forms an elevation, the torus tubarius. The tube varies in its length, and the lumen varies in its diameter. The mucous membrane lining the tube continues medially with the

Fig. 2.—A tube with an increase in the amount of glandular and adipose tissue.

mucous membrane lining the nasopharynx and laterally with the mucous membrane lining the tympanic cavity.

In a series of eustachian tubes sectioned at the Los Angeles County Hospital the mucous membrane in the cartilaginous portion was a high columnar ciliated type and in the bony portion a low columnar ciliated type. There were certain areas where the columnar cells had been replaced by squamous epithelium and areas where the columnar cells had lost their cilia. The basement membrane in the cartilaginous portion rests on the tunica propria and is structureless, while in the osseous portion it fuses with the periosteum of the bony canal. The tunica

propria is usually smooth in the bony portion but often becomes folded, thickened and redundant in the cartilaginous portion. There are more mucous glands in the pharyngeal portion and more lymphoid infiltration near the pharyngeal opening. The glands and lymphoid structure decrease as the isthmus is reached, and only a few glands generally appear in the osseous portion. In the pharyngeal portion beyond the submucous layer there is a large amount of adipose glandular tissue, which also decreases as the isthmus is reached. These evaginations or finger-like projections of the tunica propria in the pharyngeal portion sometimes may become so large as to cause obstruction of the tube and lead to permanent closure.

Fig. 3.—Direct opening (arrow) of the gland's duct into the lumen of the tube.

According to Polvogt and Babb,[4] these evaginations may extend throughout the entire length of the tube and may contain blood vessels and lymph nodules. At times they may make it impossible to pass a bougie, and they must be considered a factor in any obstruction of the tube. Semenov [5] showed a papillomatous lymphatic nodule in the bony portion of the eustachian tube, which filled most of the lumen of the tube. However, this was from an adult ear with a chronic infection. Dorothy Wolff [6] presented a case in which there was a lymphoid nodule in the

4. Polvogt, L. M., and Babb, D. C.: Laryngoscope **50**:671 (July) 1940.
5. Semenov, H.: Tr. Am. Laryng., Rhin. & Otol. Soc. **42**:563, 1936.
6. Wolff, D.: Ann. Otol., Rhin. & Laryng. **43**:483 (June) 1934.

pharyngeal portion in a child 6 months old. Lymphoid nodules are found mostly in the pharyngeal portion and may be deep in the tunica propria or on the surface of the lining membrane.

It is not uncommon in children to find large hyperplastic lymphoid structures at the opening of the tube. These undergo atrophy as the child grows older. Acute infections in the nose and throat usually involve these lymphoid structures, and instead of atrophying they become hypertrophied and cause blocking. Many authors, including Gerlach in 1875, Teutleben in 1877, Lucas in 1899 and Errecart in 1933, have stated that a tubal tonsil in the pharyngeal end of the tube is often present and may be considered a part of Waldeyer's ring.

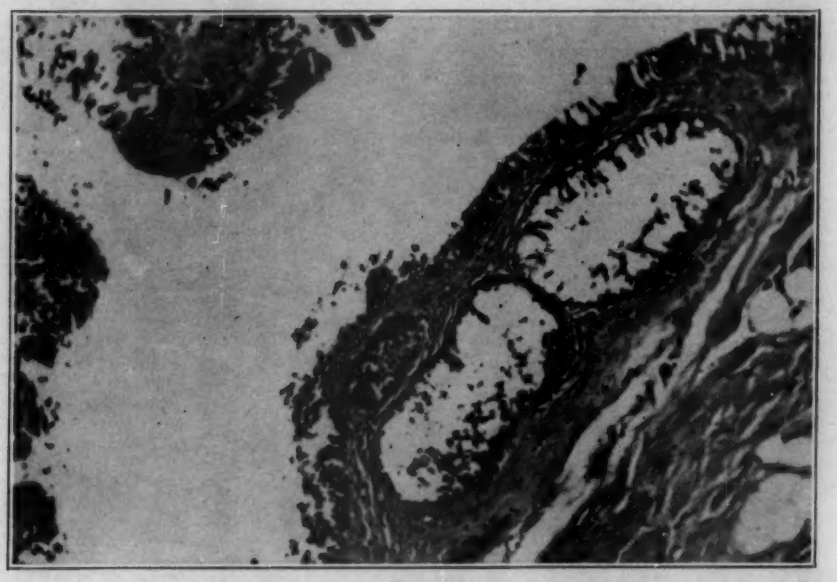

Fig. 4.—Distended glands in the tunica propria.

Beck and Guttman [7] stated that in about 28 per cent of all cases of deafness in children and in about 8 per cent in adults the condition is due to pathologic changes in the tube. Tubal involvement is bilateral in about 75 per cent of the total cases. Unilateral pathologic involvement is more frequent in adults.

Among the causes of blockage of the eustachian tube repeated attacks of rhinopharyngitis is the most common. In the acute stages of this infection the mucous membrane becomes so congested, reddened and swollen that the lumen is immediately occluded. An exudate is formed, and on examination pus is usually seen in the lumen. Generally this infec-

7. Beck, J., and Guttman, R., in Fowler, E. P., Jr.: Medicine of the Ear, New York, Thomas Nelson & Sons, 1939, pp. 205-235.

tion extends only to the isthmus, but if it goes beyond this point there are usually complications of pathologic changes in the middle ear. Histologically there is desquamation of the epithelium with loss of cilia, which even may progress to the point of ulceration. The glands in the submucous layer become enlarged and are filled with secretion. There takes place an edema of the submucous layers which may lead to suppuration. Later, after the process subsides, the glands sometimes remain swollen and enlarged for a long period. After many attacks of this kind the membranes become thickened, the glands permanently enlarged and the lumen narrowed. This all goes to produce a permanent obstruction resulting in a progressive loss of hearing.

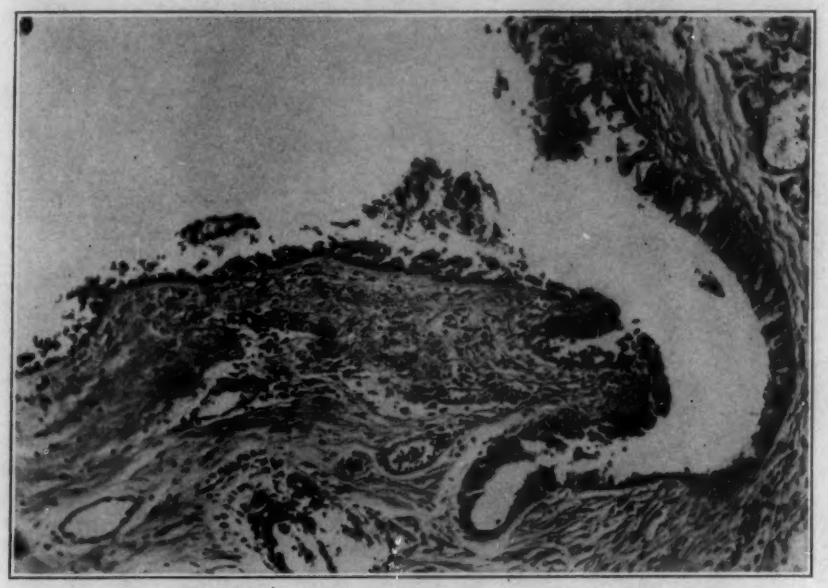

Fig. 5.—Early formation of beginning evagination.

Another important cause of tubal occlusion is hyperplasia of lymphoid tissue in the pharyngeal portion of the eustachian tube. This tissue at times becomes so heavily infiltrated and so well developed as to form large masses of so-called tubal tonsil. The adenoids themselves may become so enlarged that they exert pressure over the eustachian orifice and prevent proper opening of the tube, or they may be in such a position as directly to obstruct the orifice of the tube. Other predisposing causes of tubal obstruction may include allergic swelling and anatomic defects of the nose, adhesions and scars about the opening, tumors, neoplasms, tuberculosis, diphtheria and syphilis. Large crusts which occur in

atrophic rhinitis sometimes extend over the ostium of the tube; paralysis of the facial nerve, paralysis of the levator veli palatini muscle, hypertrophy of the posterior lips of the turbinates, rhinoscleroma, septal exostosis, calcification, ossification of the cartilaginous tube and malocclusion and overbite many times have a bearing on closure of the tube.

According to Beck and Guttman, the presence of the occluded tube has a definite influence on the middle ear. The delicate membrane of the middle ear acts similarly to the epithelium of the alveoli of the lung in its power to absorb oxygen. This results in a lower tension in the

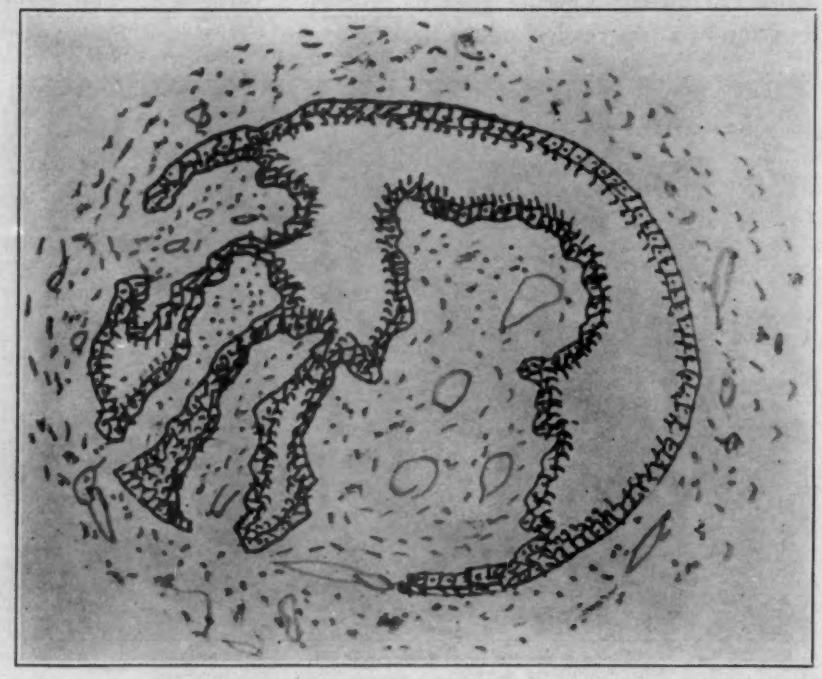

Fig. 6.—Diagrammatic illustration, showing evaginations filling most of the tube.

tympanic cavity. If the tube is in a state of continual closure there follows a decreased atmospheric pressure within the middle ear and the drum is forcibly compressed inward by the outside atmospheric pressure. This in turn interferes with the function of the sound-conducting apparatus. The excess pressure on the drum causes the patient to have a sensation of pressure, fulness and dulness in that ear. Sometimes the patient notices certain noises associated with the blood vessels and muscles of the ear which under normal conditions are not heard. These head noises and ringings constitute a tinnitus. The bone conduction of the ear is decreased, as would be shown by the tuning fork test.

The methods of physical examination of the eustachian tube are very important. They should include examination of the nasopharynx and the pharyngeal opening of the eustachian tube by means of a mirror or nasopharyngoscope, speculums or retractors, inflation of the eustachian tube in association with auscultation and if necessary bougienage, a tuning fork test by the method of Politzer and last, if necessary, roentgen examination. Digital examination may be valuable, especially for children. It may be necessary for examination of children to be done with a general anesthetic to carry out these procedures.

The treatment in the acute stages should consist of applying mild shrinking solutions to the orifice of the tube, by direct application, by

Fig. 7.—Leukocytic infiltration in early stages of inflammation of the tube.

putting drops in the nose with the head held to the side or by spraying according to the method of Pressman. The mucous membrane of the nasal pharynx should be treated locally by mild antiseptics and astringents. Any predisposing causes should be taken care of and the patient given general supportive treatment. After the acute stages are over, treatment should be in the direction of removing the cause. Any deformities or sinus trouble should be corrected. Lymphoid tissue in Rosenmueller's fossa should be eliminated. Good vision in this procedure is essential. I personally like Beck's method of retracting the soft palate. The lymphoid tissue can be removed by curet, biting forceps or adenotome. Diathermy is usually not effective on large masses of

tissue, because too much of the tissue has to be destroyed. This might be used effectively, however, for small separate follicles. Roy [7a] has suggested chemical cauterization of these follicles, but this is long drawn out and requires several applications. When the lymphoid tissue extends deep into the tube, irradiation seems to be the method of choice.

The treatment of chronic catarrhal deafness by roentgen rays has brought improvement in many cases. According to O'Brien,[8] of Boston, there was improvement in hearing and tinnitus in as high as 50 per cent of the selected patients who had received maximum roentgen ray dosage according to his technic.

Fig. 8.—High power magnification of hypertrophied glands.

Crowe and Baylor [2] used roentgen rays and radium for treatment of hyperplastic lymphoid tissue in the pharyngeal portion of the eustachian tube and nasopharynx. At present I have several children under treatment with chronic progressive deafness. After cleaning up the lymphoid tissue in the nasopharynx I have administered roentgen ray treatments to this area. Although there has been marked improvement in hearing in these cases already, it is too soon to report them at this time. Radium should be used with great care and only by those qualified to use it. Its scope of action is so small and so limited that if the lymphoid tissue

7a. Roy, D.: Frequency and Effect on Eustachian Tube of Residual Lymphoid Tissue in Larynx, Arch. Otolaryng. **24**:325 (Sept.) 1936.

8. O'Brien, F.: Radiology **28**:1 (Jan.) 1937.

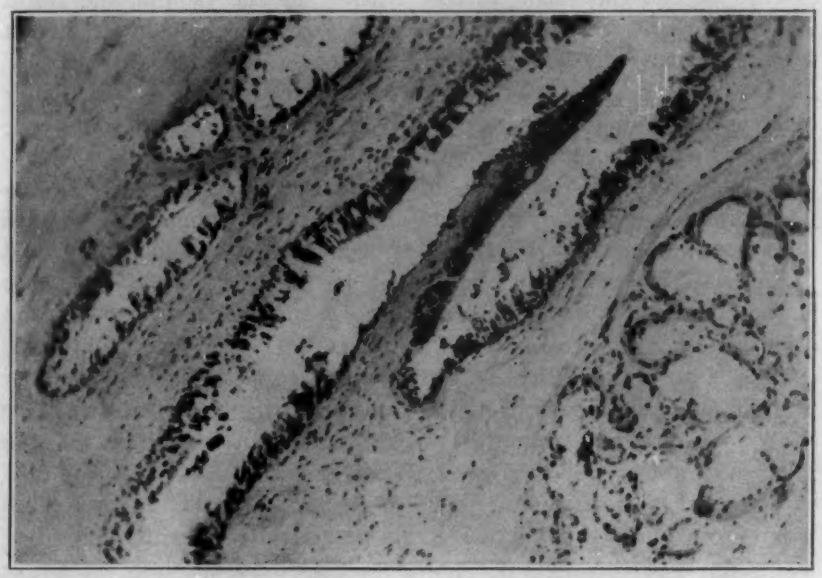

Fig. 9.—Finger-like projection which could act as valve.

Fig. 10.—Early desquamation of epithelium and piling up at the point of irritation.

extends beyond the small distance reached by the radium, the result would not be realized. For this reason, I feel that roentgen irradiation is more inclusive and generally effective than radium therapy. As for the histologic changes which take place in the lymphoid tissue after roentgen ray or radium therapy has been used, there is little difference between them, according to Schenck.[9]

Allergic swellings causing occlusions must be investigated by finding the causative agent and desensitizing the patient. Anatomic defects, such as nasal spurs and hypertrophied tips of turbinates, should be corrected surgically. Adhesions and scars about the tubal orifice can sometimes be broken up by digital massage and manipulation. Tumors and neoplasms should be treated as to their types and positions. In atrophic rhinitis large crusts should be removed and proper cleanliness applied to the nose. Obstruction of the tube due to strictures can be treated by the bougie, but great care must be taken that the mucous membrane is not injured. Simon [10] reported good results in 800 cases of pathologically involved tubes treated with the heat bougie. Instillation of iodized poppyseed oil into the tube will locate accurately the point of stricture, according to Heimendinger.[11] Malocclusion and overbite should be treated by the orthodontist. Emphasis should be placed on the prevention of colds and attention given to the patient's general health.

At present there is so much emphasis being placed on the prevention of deafness through the national health programs, hearing societies and medical committees, that it is timely to consider these factors which play such an important part in the obstruction of the eustachian tube and its effect on hearing.

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9. Schenck, H. P.: Laryngoscope **51**:780 (Aug.) 1941.

10. Simon, E.: Treatment of Chronic Catarrhal Deafness with Eustachian Heat Bougie, Arch. Otolaryng. **27**:606 (May) 1938.

11. Heimendinger, E.: Rev. de laryng. **59**:303 (March) 1938.

Case Reports

SCLEROMA

FRED W. DIXON, M.D., CLEVELAND

Within the past few years there have appeared in the literature over 100 case reports of scleroma. While most persons suffering from this chronic ailment have been foreign born, a considerable number have been native Americans. It is necessary, therefore, that this condition be kept in mind when granulation tissue appears any place in the respiratory passages.

While the diagnosis is usually a routine procedure a universal method of treatment has not been adopted. Many types of therapy have been employed. The one which at present seems to offer the best prognosis is surgical removal of the granulation tissue followed by application of radium or roentgen rays. It is earnestly desired that physicians reporting patients so treated as "improved" or "cured" state how long such improvement has been noted. It is only by observing the time element that such treatment can be evaluated.

I should like to report the following case of scleroma involving the upper and lower respiratory passages.

REPORT OF A CASE

The patient, a white man 65 years old, was born on a farm in Jugoslavia, where he lived until 21 years of age. In 1898 he came to America. Here he worked in a marble quarry for three years. In 1902 his nose began to discharge black crusts and became very dry. His breathing became more difficult and labored. In December 1902 a tracheotomy was done, but the hospital records of forty years ago have been destroyed. The tube was worn for three months. There followed a period of partial relief for two years. In 1904 a second tracheotomy was done and the tube was worn two months. At this time the trachea was dilated. A few weeks later the patient coughed and expectorated a piece of tissue, with considerable relief. From 1904 to 1913 he was employed in a foundry. For the past twenty-one years he has operated a grocery store.

He first came under my care on June 9, 1934, when he was admitted to St. Alexis Hospital complaining of cough and difficulty in breathing. His illness at that time dated to a gradual onset during the past year. His voice was husky but of fair volume. His breathing was labored. There was a dry, nonproductive cough. He was slightly cyanotic. There was no dysphagia and no loss in weight. No cervical enlargement was noted. Examination of the nose showed the breathing space on both sides to be filled with a warty granulation attached to the septum,

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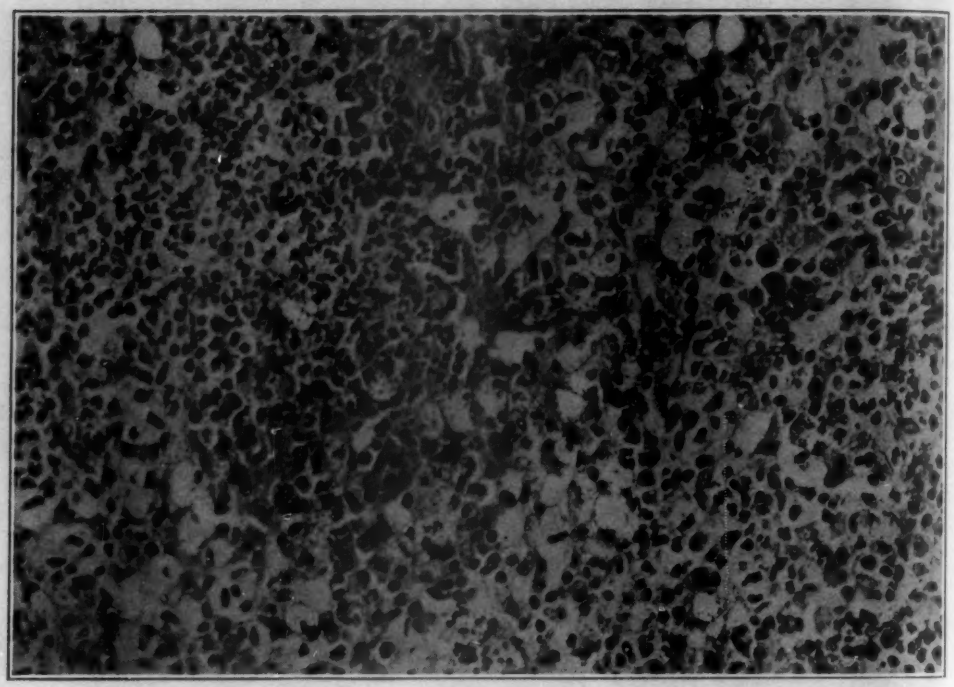
which did not shrink well on application of epinephrine. These growths were firm, did not bleed easily and were not painful. The pharynx was normal.

Examination of the trachea showed that the larynx was slightly smaller than normal. The arytenoid cartilages moved but had a limited action. Below the thickened vocal cords was a warty growth, similar to that present in the nose, which seemed to be implanted on a distorted trachea. The breathing space in the trachea was reduced. The Wassermann reaction was negative. Examination of the blood revealed nothing abnormal.

The microscopic report of the growths removed from the nose and trachea was misleading, as the interpretation was "benign adenoma." That afternoon the breathing became more labored. The patient became cyanotic, and a tracheotomy



Photomicrograph, showing granulation tissue with typical Mikulicz cells and deeply stained Unna bodies.

was performed. The lumen above the old tracheotomy wounds was reduced and fibrotic; below the wound the trachea seemed normal. He wore the tracheotomy tube for five weeks, during which time he received radiation therapy over the nose and trachea. Radium, 1,500 milligram hours, was applied on each side of the subglottic region, a total of 3,000 milligram hours. After this the larynx was dilated twice a week for six weeks. On Aug. 20, 1934 the airways seemed adequate, and treatment was discontinued.

No further trouble was experienced for four years, when, in 1938, the symptoms of obstructed airways returned. The nose was filled with granulations similar to those which had been removed, and the growth reappeared in the trachea. At this time Dr. Louis Clerf removed the tracheal growths. Exami-

nation of the tissue, at Jefferson Hospital, showed rhinoscleroma. More material was subsequently removed from the nose, and the report was similar. Klebsiella rhinoscleromatis was recovered from the tissue in pure culture on blood agar. After this the patient received radiation therapy. His breathing became normal, and he gained in weight. Bronchoscopy showed the trachea and bronchi to be free from growths. In 1941 he appeared with nasal obstruction and a recurrence of the scleromatous material in each nostril. The growths were removed surgically, and immediately afterward roentgen therapy was administered. There has been no recurrence in the nose for the past two years. The mucous membrane is moist, with slight crusting. The breathing space is adequate, although some scar tissue is forming in the choanae. The soft palate shows some retraction.

One month ago, after a quiescent period of two years, the trachea showed three small nodular growths in the subglottic space springing from the tracheal wall, which interfered with breathing. There was a small warty growth on the aryepiglottic fold. The granulations were again removed by Dr. Clerf. The patient is at present receiving high voltage roentgen therapy.

In the past eight and one-half years, during which time the patient has been under observation, operation followed by radiation therapy has been employed in the nose twice and in the trachea three times. The nose, being more accessible, seems to respond more satisfactorily to treatment than the trachea. A complete return to normal is not expected, for permanent changes have taken place, but the progress of the disease seems controlled and the patient is comfortable.

1027 Rose Building.

CHRONIC INFECTIOUS GRANULOMA OF THE NOSE

D. M. Lierle, M.D., Iowa City

I wish to present a case of so-called infectious granuloma, or progressive lethal granulomatous ulceration, of the nose.

REPORT OF A CASE

A married woman aged 22 had the onset of her present illness in 1935, when she noticed a slight ulceration in the floor of the left side of the nose in the vestibule. There was some nasal discharge, and she complained of pain in the left cheek and the upper teeth on the same side. The ulcer was about 1.5 cm. in diameter and not particularly deep, but was covered with a thick crust and was very painful. At times she had low grade fever, excessive lacrimation and headaches. In December 1936, when she was seen in the outpatient clinic, the lesion was about the same except that it was a little larger and deeper. Her past medical, family and social histories were essentially without significance. The only treatment she had received was an injection of some type by her family physician.

Physical examination revealed essentially a normal condition. Roentgenograms of the sinuses and of the teeth revealed nothing pathologic. No clinical evidence of sinus disease was present. There was no indication of blood dyscrasia, her blood count being normal in every respect. Other laboratory tests, including urinalysis, the Wassermann and provocative Wassermann tests and determinations of the blood sugar, gave normal results. The report of biopsy was chronic infectious granuloma. Smears were negative for Vincent's organism and fungi. Cultures were reported to be positive for Staphylococcus aureus haemolyticus and the beta hemolytic streptococcus.

The lesion was treated locally with boric acid compresses and later with 2 per cent yellow mercuric oxide ointment and healed within about three weeks. Between January 1937 and September 1941 the patient was seen in the outpatient clinic on three different occasions with recurrences of the ulcer, and each time the lesion responded to the simple treatment just described.

In September 1941 she again returned and reported that the nose had been all right until May of that year, when it received a mild blow. It became swollen, tender and bluish, and an abscess developed and drained into the nasal cavity. Examination revealed a greatly disfigured nose with extensive ulceration of the nasal tip and floor on the left side. There were a profuse purulent discharge, crusting and bleeding with the slightest manipulation. She was admitted to the hospital, and the disease has slowly progressed until at present there are extensive involvement of the soft tissues of the entire face and considerable exposure and destruction of the bones of the face. Secondary anemia, due to frequent small

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hemorrhages, exists. There is intense pain. The white cell count varies between 9,000 and 14,000, and the differential count remains normal. At no time has there been any evidence of adenopathy. The patient is rapidly losing weight because of her inability to eat. She has a septic type of temperature and is severely toxic. A fatal termination is inevitable within a short time.

Fig. 1.—Photographs taken (*A*) Aug. 3, 1937; (*B*) Sept. 24, 1937; (*C*) Nov. 26, 1937, and (*D*) Jan. 31, 1938.

During the present hospitalization the following procedures have been carried out:

1. Repeated general examinations by members of the section on internal medicine, with findnigs of no significance except those which one would expect with anv infection.

2. Repeated biopsies, with the same pathologic diagnosis—chronic infectious granuloma.

3. Tissue cultures, smears and animal inoculations. Staph. aureus, alpha hemolytic streptococcus, Proteus vulgaris and a diphtheroid organism were reported. Guinea pig inoculations for tuberculosis gave negative results. On one occasion an

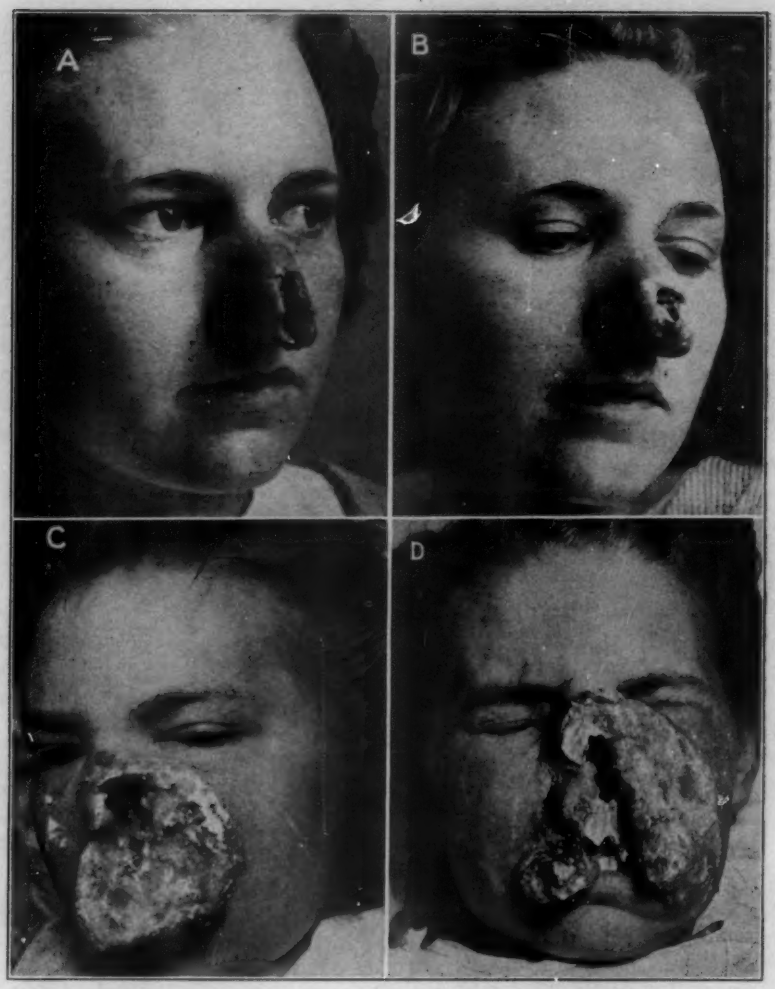

Fig. 2.—Photographs taken (*A* and *B*) Sept. 20, 1941; (*C*) March 14, 1942, and (*D*) April 27, 1942.

anaerobic streptococcus was found. Various other tests, such as determination of the blood sugar, gave normal results.

4. Cutaneous tests for sporotrichosis, with negative results.

5. Agglutination tests for typhoid, paratyphoid, tularemia, etc., with negative results.

6. Wassermann and provocative Wassermann tests, with negative results.

7. Frei tests for lymphogranuloma venereum, with negative reactions with the human antigen.

8. The Strauss test for glanders, with a negative reaction.

The following treatment was carried out, most of it in desperation, during this stay in the hospital:

1. Local applications of boric acid dressings and various ointments and dye antiseptics.

2. Administration of sulfanilamide, sulfathiazole (2-[paraaminobenzenesulfonamido]-thiazole) and sulfadiazine (2-[paraaminobenzenesulfonamido]-pyrimidine).

3. Roentgen irradiation (400 r).

4. Administration of mapharsen.

5. Routine antisyphilitic therapy (department of syphilology).

6. Fever therapy.

7. Repeated transfusions, intravenous injections and other supportive measures.

8. The Straus test for glanders, with a negative reaction.

9. High vitamin therapy.

The laboratory on one occasion, about six months ago, reported the presence of an anaerobic streptococcus, and I have been using zinc peroxide up to the present. At first there was decided improvement, but lately the disease has progressed in spite of the treatment.

My use of zinc peroxide was based on the success Meleney has had with it in cases of chronic necrotizing ulcers seen in general surgical practice. He has shown that the beta hemolytic streptococcus which is present in these lesions is a facultative anaerobe, or, as he calls it, the microaerophilic hemolytic streptococcus. This, combined with Staph. aureus haemolyticus, may cause ulcerative lesions. He has been able to reproduce in animals lesions similar to those found in human beings.

Several months after the last admission of the patient, although the disease was far advanced, radical surgical intervention was contemplated, but, while the patient was willing, there were other objections and this procedure was not carried out.

This case is presented with the hope that some one may have suggestions in regard to the etiology and treatment of this disease. Early recognition, with radical surgical measures, offers, in my opinion, the only chance at present of doing anything for a patient with this condition.

University Hospitals.

INHALATION OF A FOREIGN BODY

Report of Two Unusual Cases

LOUIS M. FREEDMAN, M.D., BOSTON

The following cases are reported because of unusual conditions in relation to them. The first case is that of a foreign body, a small three stone ring, in the esophagus of a child, the presence of which was unrecognized for more than six years. The second case is interesting because of the confusion caused by the presence of a foreign body in the external tissues, which was discovered at the time another foreign body was inhaled.

REPORT OF CASES

CASE 1.—A girl aged 7 years was admitted for tonsillectomy to the otolaryngologic service of the Boston City Hospital on Feb. 10, 1942, with the diagnosis of chronic hypertrophied tonsils. A routine preoperative roentgenogram of the thymus failed to show enlargement of the gland but did reveal a "ring-shaped shadow in the midesophagus." There was no history of difficulty in swallowing or breathing and no evidence that the presence of the ring in the esophagus was known either to the child or to her parents.

Closer questioning of the parents elicited the fact that at the age of 6 months the child had been presented with a small three stone ring, which had promptly disappeared, and no evidence of its whereabouts had been discovered. Since that time the child had been ill with measles, whooping cough and chickenpox and had recovered without special incident. It was difficult to reconcile this symptomless history with the presence of the ring in the esophagus for more than six years. Further questioning brought out the fact that on June 26, 1940, approximately twenty months before the present admission, the child was brought into the hospital with bilateral Colles fracture and at that time a roentgenogram of the thymus was negative for enlargement of the gland but revealed a "probable extrinsic metallic ring overlying the supracardiac area." In the excitement of an accident floor reduction of bilateral Colles fracture, with the patient under general anesthesia, the foreign body in the esophagus received no attention.

From this definite history and roentgenographic evidence, it seemed plausible that the ring must have been present in the esophagus since its early disappearance, when the child was 6 months old. It became necessary, then, to explain how this ring could remain in a position which under ordinary circumstances did not represent any of the normal narrowings of the esophagus. Only one reasonable explanation fitted this picture. It was necessary to assume that at the age of 6 months the lumen of the esophagus was, if anything, slightly narrower than the diameter of the ring. The ring must have impinged on the membrane so as to remain comparatively fixed in spite of deglutitory efforts. Furthermore, it probably excoriated the mucous membrane of the esophagus, which caused the formation of granulations and fibrous tissue, with eventual fixation of the ring in the esophagus at this level. It was evident that at the age of 7 years the

lumen of the esophagus was almost twice the diameter of the ring and only fibrous tissue could hold the ring in place; otherwise, it would have passed down through the cardia.

Three days after admission the patient was given ether anesthesia, and, with a Roberts 30 cm., 12 by 16 mm. esophagoscope, the ring was visualized at the junction of the upper and the middle third of the esophagus. It was fixed in place by adhesions in the right anterolateral region. It occupied about one half of the diameter of the lumen of the esophagus and by surface tension lay on the posterior wall. With grasping forceps on the ring, one could lift the whole esophagus, making it evident that the adhesions about the ring were fibrous.

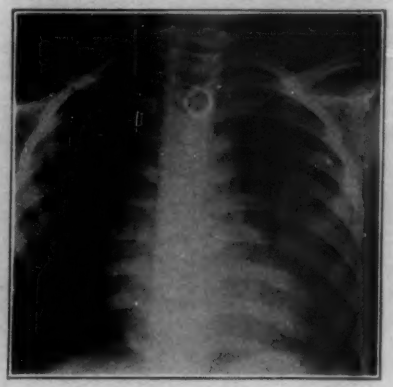

Fig. 1 (case 1).—Roentgenogram, showing presence of ring in the esophagus.

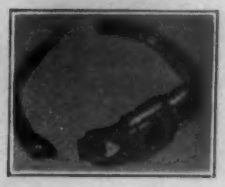

Fig. 2 (case 1).—Photograph of ring after removal.

The question then arose as to the best method of removal. The fibrous tissue band was tightly adherent to the ring, although the ring could be turned anteriorly by its free portion, held only by the fibrous band. It was deemed undesirable to cut the adhesions for fear of injury to the esophagus. The ring was therefore cut with a caliper rotation forceps in two places, one in front of and one behind the portion containing the stones, and the two parts were removed separately. The portion with the stones was loose, and the ring portion was turned out of the space created by the fibrous band and the esophagus.

The postoperative course was uneventful. Two weeks later the tonsillectomy for which the child entered the hospital was performed. The patient was discharged without incident.

Case 2.—A seamstress in her early twenties, whose habit it was to keep pins in her mouth preliminary to basting, came into the hospital in the early evening

with a history of having inhaled a common pin. The roentgenogram showed two pins rather than one—one in the upper portion of the pharynx, which was removed, the other in the region of the upper lobe of the left lung. Bronchoscopic examination that night failed to reveal the presence of a pin in the trachea or bronchi. The next morning closer study of the roentgenogram and fluoroscopic observation of the foreign body with the patient's left arm lowered and raised revealed that the remaining foreign body was not in the lung at all, first, because it was above the level of the bifurcation and the trachea was medial to it; second, because, with the upper end of the foreign body directed more medially than the lower, it was impossible for it to have been inhaled into the upper lobe and have assumed that position, and, third, because when the patient's arm was raised and lowered there was a wide excursion of the foreign body of about 3 inches (7.5 cm.), which would have been impossible if it had been in the lung.

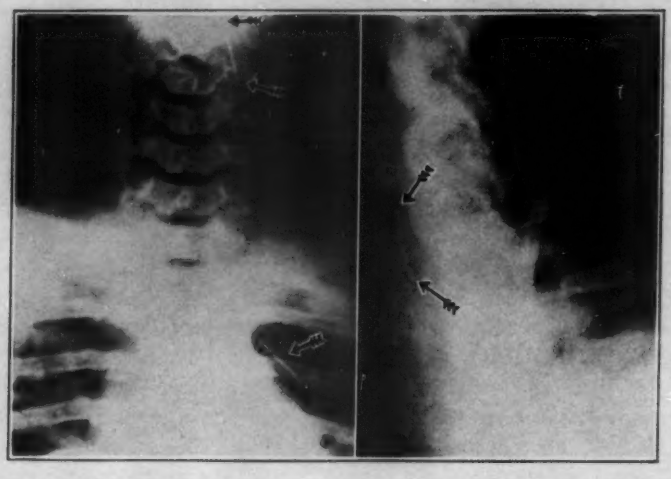

Fig. 3 (case 2).—Roentgenograms, showing presence of a foreign body in the musculature of the back.

Further, the body was observed to be posterior rather than anterior and was definitely placed in the musculature of the back, just behind the scapula. Finally, closer examination of the object showed it to be not a common pin but the beveled end of a hypodermic needle. A history elicited later revealed that a needle had been broken when a hypodermic injection was given in that region several years earlier, in the patient's childhood, and no attempt had been made to remove it, since it caused no symptoms.

Because the two objects were similar in appearance, it was natural to assume that both were inhaled at the same time, just before the present admission to the hospital. This case demonstrates the importance of more exact study before undertaking repeated bronchoscopic examinations, even though an immediate bronchoscopy at the time of entry, before the foreign body had advanced too far, might be entirely justified.

475 Commonwealth Avenue.

Abstracts from Current Literature

Ear

FENESTRATION OF THE LABYRINTH IN CHRONIC CONDUCTIVE DEAFNESS: AN ANALYSIS OF RESULTS OBTAINED. EDWARD H. CAMPBELL, South. M. J. **34**:13 (Jan.) 1941.

Campbell calls attention to the gratifying results being obtained in the surgical treatment of chronic progressive deafness. He credits Jenkins, Bárány, Holmgren and Sourdille with being the pioneers in this work and states that Lempert has modified Sourdille's technic into a practical one stage operation. The surgical procedures for the treatment of chronic progressive deafness have as a basis the creation of a fenestra in some part of the labyrinth of the inner ear, and the real problem in attempts to advance their practicability has been to devise means of making the fenestrated labyrinth remain permanently open.

To obtain good results it is important to select the patients properly. The patient should have conductive deafness with a loss of practical hearing that is not too profound. The bone conduction should be good. Increased bone conduction, a negative Rinne test and a Weber test lateralizing the sound to the bad ear indicates favorable bone conduction. The vestibular mechanism should be normal as shown by the Bárány and caloric tests. There should be no aural infection. The tympanic membrane and the middle ear should preferably be normal. The membranous and bony canal walls should be normal. The patient's general condition should be good.

The hearing result should not be judged as final until at least one year after the operation. The results obtained in fenestration operations in this country in the past three years have been impressive and should stimulate wider use of the procedure.

The author states that a favorable result is obtained when there has been a permanent improvement of the hearing to a practical level, a lessening of the tinnitus, or a prevention of further progression of the deafness. In the most favorable cases an improvement of 30 to 35 decibles may be obtained in the three conversational frequencies (512, 1024 and 2048). However, in the majority of successful cases the average gain is 18 to 20 decibles, and in the less successful cases the gain may be only 8 or 10 decibles, in the conversational frequencies.

The author has operated on 31 patients but only 17 had the proper prerequisites for good results. Of the 17 having the proper prerequisites 14 retained satisfactory hearing, giving a success percentage of 82.4. Of the 17 who have retained improvement from the operation, 8 were operated on a year or more ago and have retained an average improvement of 17.4 decibles for the three conversational frequencies; the remaining 9 show an average improvement of 21.7 decibles.

In its present state of development the operation for fenestration of the labyrinth is definitely a practical procedure for the improvement of hearing in properly selected patients with chronic progressive deafness. With the experimental and clinical work now in progress, rapid advancement in the use of this technic is certain to follow in the near future.

SINGLETON, Dallas, Texas.

CAUSES OF FAILURE IN THE TREATMENT OF ACUTE OTITIS MEDIA: A DISCUSSION. T. B. LAYTON AND OTHERS, Proc. Roy. Soc. Med. (Sect. Otol.) **34**:225 (Feb.) 1941.

The vast majority of patients with otitis media, Layton held, get well by themselves, and chronic otitis media is common only in communities where nutrition is deficient both from the caloric and from the vitaminic point of view.

He thought paracentesis should not be done in the acute invasive stage, that antiseptics in the external ear were of little value, that it was important to prevent recurring infections in the nasopharynx, that the prevention and the cure of otitis media were encouraged by an erect posture of the child, that removal of adenoids during the disease was improper treatment, that activity in the patient should be encouraged to increase the circulation, and that mastoidectomy should be considered if the ear is not well in six weeks.

Mr. E. Musgrave Woodson thought that failure in the treatment was due to presence of complications, such as persistent suppuration in the eustachian tube, unsuspected involvement of the mastoid, invasion of the ossicles and presence of the tubercle bacillus.

Mr. J. Douglas McLaggan advocated paracentesis only when pus was present in the middle ear.

Mr. F. C. Ormerod thought paracentesis should be done when the color of the drum had changed from red to blue-gray and the bulging had spread over the handle of the malleus.

Mr. Terrence Cawthorne thought the causes of failure were neglect in the early stages to apply the general principles for treatment of inflammation and to the presence of an infection greater than the resistance of the patient.

In closing Mr. Layton maintained that the diminution in virulence of the hemolytic streptococcus came before the advent of chemotherapy and that it was likely to become virulent again.

CAMPBELL, Philadelphia.

THE LIMITATIONS OF OPERATIVE TREATMENT IN TRAUMATIC FACIAL PARALYSIS: A DISCUSSION. D. J. COLLIER and others, Proc. Roy. Soc. Med. (Sect. Otol.) **34**:575 (July) 1941.

When the facial nerve is damaged in a mastoid operation, conductivity, according to Miss Collier, may be lost either by pressure from the outside which prevents the passage of the nerve impulse or by complete section of the nerve or actual loss of substance. The pressure may be simply that of edema or hemorrhage with an intact sheath, or a torn sheath, or it may be that of detached bone from fracture of the wall of the fallopian canal. Exploration alone can determine whether the nerve has been divided or merely compressed, and should be undertaken if in the third week after onset of the paralysis examination with the patient under general anesthesia shows the reaction of degeneration and absence of faradic response. Operative damage to the facial nerve may involve the segment in the petrous bone, that in the middle ear at the bend below the external semicircular canal or that in the descending portion. As power cannot be restored to muscles which have undergone fibrous atrophy, absence of galvanic responses is a contraindication to any operative treatment directed to the nerve supply.

Nerve grafting in the temporal bone, Miss Collier said, owed its success to the presence of the rigid bony channel in which the inlay could be maintained without sutures. Minimal formation of scar tissue was insured by accurate approximation of nerve and graft. This required a graft equal in caliber to the recipient, which for the facial nerve could be supplied by the middle cutaneous nerve of the thigh. To secure perfect apposition, the fixed ends of the graft and the nerve must be cut accurately at right angles to their long axis. This is more easily done with the degenerated graft.

Mr. J. Z. Young stated that the rate of growth of nerve fibers through fresh and through predegenerated autografts had been measured in rabbits. No appreciable differences were found, and any advantage of the predegenerated grafts must therefore be due to other factors, such as consistency and ease of manipulation. He thought the placing of facial grafts might be assisted by the use of the concentrated plasma solutions described by Medawar and Young (*Lancet* **2**:126, 1940).

Dr. F. S. Cooksey said that usually the electrical changes of the reaction of degeneration did not appear before ten days after the injury and were not reliable

before the fourteenth day. Failure of the nerve to respond to the full strength of the faradic coil, together with the sluggish galvanic response in the muscle, could be taken as strong evidence that the reaction of degeneration was complete. Some patients will not tolerate sufficient faradic current for adequate clinical examination, but this difficulty can be overcome by repeating the examination with the subject under an anesthetic, such as pentothal sodium.

He further stated that galvanic stimulation of the paralyzed muscle might be harmful to the muscle and delay recovery. He thought it should be used to produce about ten gentle contractions of each muscle daily. Recovery may continue for three or four years after nerve grafting, but usually the maximum is reached in two years. Complete dissociation of the facial movement is seldom obtained.

Mr. Terrence Cawthorne and Mr. A. Dickson Wright gave further opinions on the methods of nerve grafting, after treatments, etc.

CAMPBELL, Philadelphia.

THE AURICLE IN THE FUNCTION OF SOUND LOCALIZATION. L. CHAVANNE, Oto-rhino-laryng. internat. **25**:97 (July) 1941.

The experiments of Gellé clearly indicated the function of the auricle in collecting and locating sounds. They were incomplete so far as localization is concerned, since the observations were limited to a single ear. The direct zone of audition may be represented as the area of a triangle with sides of 50 or 60 degrees, whose open angle is directly forward from the subject; laterally on each side lie the zones of mixed audition; the zone of indirect audition lies to the rear. From the direct zone all sounds reach the ear without reflexion or diffraction. Sounds arising in the zones of mixed audition are located by two procedures utilizing the difference of intensity and the difference in phase of the two ears. The first is used for acute sounds, which are reenforced by the sides of the head and the auricle; for low-pitched sounds, determination of the difference of phase perceived by the two ears is required. There is an intermediate zone, between c^3 and g^4, for which both procedures are needed. Sounds arising in the direct and in the indirect zones are localized by the absence of difference of intensity and of phase. To differentiate between sounds arising in one of the two mixed zones, the aid of vision and of rotating the head is necessary. The localization of high or low tones in height (vertically) is allied to the form of the head and of the auricle.

DENNIS, San Diego, Calif.

PRIMARY LATENT MASTOIDITIS WITH FACIAL PARALYSIS. L. LOPEZ VILLORIA, Arch. venezol. Soc. de oto-rino-laring., oftal., neurol. **2**:5 (March) 1941.

Villoria presented a case of right-sided complete facial paralysis, apparently peripheral in nature. The patient was 15 years old. The paralysis apparently occurred spontaneously, without any frank cause, two months before examination. There was no history of nasal catarrh, grip or infection of the throat, nor of exposure to cold. The physical examination gave essentially negative results. The ears and mastoids appeared grossly normal.

Roentgenologic study of the mastoid processes in the ordinary positions of the head revealed well defined clearcut pictures on both sides, without any evidence of pathologic change. However, in altering the position of the head into a transverse oblique direction, which would permit the temporal tympanic region to come clearly into view, a small area of density appeared posterior to the external auditory canal, in front of the knee of the lateral sinus and on the side posterior to the angle of Citelli. The cells were obscured, and part of the intercellular septums had disappeared.

Throughout the course of observation the temperature of the patient was never above 37.5 C. (99.5 F.). Myringotomy was performed, but there was no pathologic secretion. The patient was given sulfapyridine (2-[paraaminobenzenesulfonamido]-pyridine) 4 tablets a day. This was later reduced to 3 tablets a day.

A subsequent roentgenogram revealed complete clearing up of the pathologic process in the mastoid. It was taken about two weeks after he was first observed by the author. At that time, too, all evidence of the paralysis had disappeared, and the youth appeared to be in perfectly normal health.

Because there was no clinical evidence of mastoiditis other than the haziness of the mastoid process somewhat behind the antral region, Villoria felt that this focus of change was responsible for the facial paralysis. In view of the absence of clinical symptoms, he decided to term this case one of latent primary mastoiditis. This is purely on roentgen evidence. One interesting point was stressed, namely, that when the diagnosis is obscured and the ordinary roentgenologic views are inconclusive, it is most important to have a second roentgen examination with the position of the head altered so that some small obscure area of infection or change may be brought out more clearly. He stresses the temporotympanic plane of Schüller, Law and Cottenot and the occipitozygomatic plane of Arcelin and Stenvers.

He believes that it was due to the sulfapyridine that a rapid and complete cure was effected and mastoidectomy obviated. PERSKY, Philadelphia.

DOUBLE OTOGENIC ABSCESS OF THE BRAIN, WITH HEALING. W. SPRENGER, Monatschr. f. Ohrenh. **75**:255 (May) 1941.

The author reports a case of acute otitis media in a physician which progressed to formation of a brain abscess despite good clinical observation and treatment. After the first abscess in the temporal lobe was opened the symptoms increased and intracranial pressure persisted, and a second abscess cavity was discovered. This abscess had no connection with the first. As the result of daily changes of iodoform dressings, both cavities healed, and the patient recovered completely. The self observation of this patient made the timing of the duration of the abscess simple. The patient could establish the time of the initial stage, in his case an epileptiform attack. The latent period lasted about sixteen days, inasmuch as from the seventeenth to the thirty-fifth day vague intracranial symptoms were already present, which finally led to the manifest stage. It is remarkable that during this short time two abscess cavities 8 and 6 cm. in diameter, respectively, could develop.

LEDERER, Chicago.

THROMBOSIS OF THE PETROSQUAMOUS SINUS. O. NOVOTNY, Monatschr. f. Ohrenh. **75**:260 (May) 1941.

In a patient operated on as the result of symptoms of thrombosis of the sigmoid sinus a vein was found above the antrum, close to the dura, leading into the upper knee of the sinus. This vein was completely thrombosed. The sigmoid sinus itself was normal. It can be assumed that this vein was a persistent petrosquamous sinus. The author presents several photographs of an anatomic specimen to support the description of the course of this sinus. Occasionally roentgenographic studies might reveal its presence. The necessity for considering the possibility of thrombosis of the petrosquamous sinus in an unclear picture of otogenic complication is stressed.

LEDERER, Chicago.

Pharynx

TULAREMIA WITH EXTENSIVE PHARYNGITIS. ROBERT R. ANSCHUETZ, Am. J. Dis. Child. **62**:150 (July) 1941.

This is the report of a case of tularemia in which the chief clinical manifestation of the disease was a necrotizing ulceration of the entire pharyngeal wall, extending to the surface of the tongue and to the oral mucosa. The author believes that this ulceration represents the point of entry of the organisms into the body. He refers to 3 similar cases in the literature.

NELSON PAUL ANDERSON, Los Angeles [ARCH. DERMAT. & SYPH.].

INDICATIONS FOR TONSILLECTOMY. GEORGE M. COATES and WILLIAM GORDON, Pennsylvania M. J. **45**:218 (Dec.) 1941.

The authors devote a considerable portion of their paper to a discussion of the "focus of infection, the anatomy, the function of the tonsil and the examination." They state that while a healthy tonsil has one or more functions in early life, an infected tonsil loses a large part of this function and may therefore actually encourage further local and systemic infection. It is admitted that in spite of a most careful examination of the tonsils, the examiner cannot be sure that infection is not present.

Indications for tonsillectomy are placed under these headings: (1) conditions within the tonsils; (2) conditions adjacent to the tonsils; (3) conditions remote from the tonsils.

In the first group are repeated attacks of tonsillitis, abscesses within the tonsils, epidemic streptococcic sore throat, tuberculous involvement of tonsils, diphtheritic involvement of tonsils (carriers), hypertrophy of tonsils, Vincent's infection of the tonsils and halitosis due to tonsillar infection.

In the second group are acute catarrhal and suppurative otitis media, chronic suppurative otitis media (under certain conditions), middle ear deafness, labyrinthitis and middle ear deafness, sinusitis, thyroid disease, laryngitis, pharyngitis, cervical adenitis, peritonsillar abscess, ocular diseases, acute phlebitis and thrombophlebitis of the vessels of the neck.

In the third group are listed a large number of remote systemic diseases which can result from tonsillar infection. WEINBURGER, Sayre, Pa.

NEUROLOGIC SYMPTOMS ASSOCIATED WITH MALIGNANT TUMORS OF THE RHINOPHARYNX. ERIK GODTFREDSEN, Acta psychiat. et neurol. **16**:47, 1941.

Twenty-five (40 per cent) of a series of 64 patients with cancer of the rhinopharynx had neurologic symptoms. The commonest symptoms were those referable to involvement of the cranial nerves at the base of the brain within the skull. Of particular interest were 8 patients in whom trigeminal neuralgia developed early in the course of the disease. In every case in which trigeminal neuralgia appears early the patient should be specifically examined for the presence of cervical lymphadenopathy, which almost invariably occurs with tumors of this type. Extensive and intensive roentgen irradiation of the involved regions is the treatment of choice. Of the 25 patients reported on, 3 were free from symptoms six to ten months, 2 two to two and one-fourth years and 5 six to eight and a half years after treatment. BRENNER, Boston [ARCH. NEUROL. & PSYCHIAT.].

Larynx

CLINICAL CONDITIONS ARISING FROM ANOMALIES OF MALDEVELOPMENTS OF THE BRANCHIAL ARCHES OR CLEFTS. WALTER B. HOOVER, Ann. Otol., Rhin. & Laryn. **50**:834 (Sept.) 1941.

Hoover reports on material gathered largely from the Lahey Clinic with respect to (1) variations in the right recurrent laryngeal nerve and the great vessels in the upper portion of the chest and the neck; (2) epidermal inclusion (dermoid) cysts; (3) preauricular sinuses, supernumerary cartilages about the ear and maldevelopment of the auricle, external auditory meatus and middle ear; (4) lateral cervical branchiogenic cysts and sinuses, and (5) anomalies of the thyroglossal tract and aberrant thyroid. These conditions are all briefly discussed and the management discussed. The direct innervation of the larynx from the vagus nerve by way of the inferior laryngeal nerve on the right side and the anomalous development of the right subclavian artery from the arch of the aorta on the left, so that it crosses behind the esophagus and presses on it, occur with sufficient frequency that laryngologists should keep the possibility in mind.

M. V. MILLER, Philadelphia.

BRONCHOSCOPY IN THE TREATMENT OF POSTOPERATIVE PULMONARY ATELECTASIS. WILLIAM A. LELL, Pennsylvania M. J. **44**:1551 (Sept.) 1941.

In the discussion of the modus operandi of the development of postoperative atelectasis a distinct similarity to that of collapse of the lung due to occlusion by a foreign body is shown. The etiologic factors in production of postoperative atelectasis are listed as follows: (1) accidental aspiration of food or secretions during the operation; (2) recent infection of the upper respiratory tract; (3) any unsuspected pathologic process of the lung; (4) unrecognized bronchiectasis; (5) indiscriminate use of postoperative sedation; (6) the too frequent, improper and often unnecessary use of intratracheal anesthesia.

Massive atelectasis is associated with such startling symptoms as sudden rise in the temperature, pulse and respiratory rate and respiratory distress, so that the diagnosis is apparent. For the diagnosis and differential diagnosis of the milder types, roentgenographic examination is strongly advocated. In the differential diagnosis, postoperative hemorrhage, pulmonary embolism or infarction, coronary disease and subdiaphragmatic abscess are to be ruled out.

In the treatment, the author feels that bronchoscopy is not advisable in all cases of postoperative atelectasis. The milder forms often clear spontaneously under a definite routine of palliation, such as use of a snug binder, hyperventilation (ten minutes hourly) and postural drainage. Morphine or atropine is not to be administered. If these measures fail, bronchoscopic examination is carried out. In cases of massive atelectasis this procedure should be performed without delay.

WEINBERGER, Sayre, Pa.

ACUTE LARYNGOTRACHEAL BRONCHITIS: CURE BY ENDOSCOPIC THERAPY. M. GONZÁLEZ LOZA, Rev. otorrinolaring. **1**:11 (Sept.) 1941.

González Loza discusses the histopathology, symptomatology, diagnosis and treatment of acute laryngotracheal bronchitis in children. He presents 2 cases in detail in which cure was effected with endoscopic treatment. He concludes: 1. Acute laryngotracheal bronchitis is a clinical entity. 2. It is both a contagious and a seasonal infection, the appearance of which is often coincident with epidemics of grip and diphtheria. 3. The disease occurs relatively constantly in the part of Argentina with which the author is familiar, but it is often unrecognized by the internist and the pediatrician. 4. The condition is easily confused with other acute infections of the respiratory tract. The internist can make only a tentative diagnosis, but the directoscope provides the one method by which one can make the diagnosis with certainty. 5. Endoscopic examination should be obligatory in all cases of suspected laryngotracheal bronchitis, especially in children. 6. The therapeusis in this disease is essentially of a mechanical order and should be directed toward the obstructive factor. Aspiration of the secretion with or without the instillation of a cajuput oil from Melaleuea viridi flora (gomenol) is the method of preference. 7. Bronchoscopy has fundamentally altered the prognosis in this disease. Whereas formerly there was a mortality of 100 per cent, many cures are now effected, especially when the patient is seen early and there is no opportunity for extensive destruction of the tracheobronchial tree.

PERSKY, Philadelphia.

CHANGES IN INDICATIONS FOR TRACHEOTOMY. H. FUCHS, Monatschr. f. Ohrenh. **75**:208 (April) 1941.

In connection with the case of a patient suffering from laryngeal phlegmon which necessitated an emergency tracheotomy, the author discusses the historic background of this operation. Two basic principles in the treatment are present: first, provision for free inspiration and expiration through the trachea and, second, the creation of an adequate access to the air passages below the level of the

glottis. The author believes that an indication for either need not constitute an emergency. It will depend on the conduct of a given case whether or not a prophylactic tracheotomy will suddenly develop into an emergency operation. An enumeration of indications for tracheotomy follows, including foreign bodies, inflammatory changes of an acute or chronic nature, pressure from without, pareses and neoplasms, and, finally, it may serve as a preparatory measure for other operations. In general, it may be stated that no tracheotomy need be performed in cases of foreign bodies because of the widely developed use of direct laryngeal and tracheal examination. Only in 3 cases in fifteen years at the Vienna clinic was tracheotomy necessary for removal of a foreign body. These operations occurred as emergencies, inasmuch as in 1 instance the foreign body could not be dislocated in sufficiently short time and in another, the case of an infant, edema followed the removal of a foreign body. In cases of trauma, in addition to the damage arising from the primary injury, the secondary danger of hematoma, edema, emphysema or dislocation of the cartilaginous fragments frequently makes an emergency operation necessary. The widest range of usefulness for emergency tracheotomy is to be found in inflammatory processes in the larynx, especially those edematous reactions of the loose submucous tissue producing edema of the larynx as a result of trauma, stasis, collateral edema or an angioneurotic or toxic condition.

LEDERER, Chicago.

Nose

EXPERIMENTAL SINUS SURGERY: EFFECTS OF OPERATIVE WINDOWS ON NORMAL SINUSES. ANDERSON C. HILDING, Ann. Otol., Rhin. & Laryng. **50**:379 (June) 1941.

The experiments were carried out on the maxillary sinuses of rabbits. The results show that after window operations active suppurative sinusitis may develop even though the sinus was normal at the time of operation. The results seem to depend on the position of the window with reference to the ciliary action of the intrasinal mucosa. Windows made near or at the ostium seemed to predispose to suppuration, but if windows were made at a point distant from the ostium suppuration did not occur. It is felt that, as the ciliary streams from the entire sinus cavity are directed toward the ostium, interference with this action is the cause of the suppuration. Windows made at intermediate points sometimes did and sometimes did not cause suppuration. Wide open windows, as well as obstructing scars, can interfere with ciliary action.

M. V. MILLER, Philadelphia.

A BROADER CONCEPT OF FUNCTIONAL DISEASES OF THE NOSE. C. STEWART NASH, Ann. Otol., Rhin. & Laryng. **50**:458 (June) 1941.

One must realize the relation between the function of the nasal mucosa and the function of the nose itself. Briefly, the function of the mucosa is to produce mucus, but it must be produced within certain quantitative limits for perfect maintenance of the function of the nasal mucosa. Any shifting of this production of mucus to either hyperfunction or hypofunction will result in a functional nasal disturbance, the signs and symptoms depending on the amount and character of the dysfunction and the length of time it has continued. The author briefly reviews the conditions producing hypofunction and hyperfunction, pointing out the fact that physical agents, such as temperature and humidity, as well as psychic states, foods, drugs, infections and endocrine imbalance all may produce changes in nasal function and that more careful history taking and investigation will lead to better diagnosis and more efficient treatment.

M. V. MILLER, Philadelphia.

MANAGEMENT OF CAVERNOUS SINUS THROMBOPHLEBITIS. GORDON D. HOOPLE and IRL H. BLAISDELL, Ann. Otol., Rhin. & Laryng. **50**:503 (June) 1941.

The discussion is confined to the anterior fulminating type of thrombophlebitis of the cavernous sinus. It is usually due to infection of the sinus through the ophthalmic veins, occasionally by way of the pterygoid plexus. The acute fulminating type differs considerably from the "chronic compensatory" type, which results from retrograde thrombosis from vessels communicating with the cavernous sinus. In many cases of the latter type there is only thrombosis, and not thrombophlebitis, and the clot may be sterile. Most cases of the fulminating type follow lesions about the upper lip and nose, and the organism is usually the staphylococcus. The criteria for diagnosis are: (1) a known site of infection; (2) evidence of infection of the blood stream; (3) early signs of venous obstruction in the retina, conjunctiva or eyelid; (4) paresis of the third, fourth and sixth nerves, resulting from inflammatory edema; (5) formation of an abscess in the neighboring soft tissues, and (6) evidence of meningeal irritation.

The authors report 2 cases, in 1 of which the patient recovered. They plan the future management of patients accordingly: (1) Start treatment early; (2) discard ligation; (3) seek ophthalmologic consultation early and often, even as often as every three hours; (4) determine the causative organism and give the appropriate drug of the sulfanilamide group (if the causative organism cannot be determined, use sulfathiazole (2-[paraaminobenzenesulfonamido]-thiazole), as most infections are staphylococcic); (5) with the drug give heparin, in large doses at first to reduce the coagulation time as soon as possible and then in regulated doses to maintain a two hour level for the coagulation time; (6) watch the blood, urine and spinal fluid for evidence of infection and treat accordingly, and (7) operate if patient's progress is unsatisfactory and surgical treatment is indicated.

M. V. MILLER, Philadelphia.

SINUS INFECTIONS: THEIR RELATIONSHIP TO RESPIRATORY DISEASE. FERRIS ARNOLD, California & West. Med. **54**:270 (May) 1941.

Operations on the sinuses should rarely be performed on children. Tonsils and adenoids should not be removed during the course of an acute infection; in the presence of chronic or subacute infection, however, they are often the site of residual infection and should then be removed. Infections of the nasal accessory sinuses are important etiologic factors in respiratory disease. It is a mistake to assume that any vaccine or protein injection treatment will cure the sinus infection unless proper drainage has been obtained and foci of infection have been removed.

DAVIS, San Francisco [AM. J. DIS. CHILD.].

ASPECTS OF SINUSITIS: THE TREATMENT OF CHRONIC ETHMOIDITIS BY THE TRANSANTRAL OPERATION: A DISCUSSION. J. C. HOGG AND OTHERS, Proc. Roy. Soc. Med. (Sect. Laryng.) **34**:117 (Jan.) 1941.

J. C. Hogg discussed a series of cases in which chronic ethmoiditis had been treated surgically by the transantral route. All the patients presented signs of severe sinusitis with purulent discharge and polypoid degeneration of the mucosa in the middle meatuses of the nose. It was difficult, he said, to achieve complete exenteration of the ethmoidal cells even by the external operation, and he abandoned the intranasal for the transantral route. Operation was best performed with the patient under general anesthesia. The Caldwell-Luc operation is performed first, then the ethmoid cells are exenterated and finally the sphenoid sinus is opened. The middle turbinate can be removed or left in, according to its condition. He stated that 132 patients had been treated by the transantral operation, and 106 of them had been inspected at least twelve months after operation; of these, 59.4 per cent had healed clean noses and were free from nasal symptoms, while 26.4 per cent had residual infection. He concluded that this operation offers a fair prospect for a lasting cure of ethmoiditis and that it possesses many advantages.

S. E. Birdsall performed an operation of a similar nature on 50 patients, most of whom had had previous intranasal operations.

W. J. Daggett appeared to be more partial to the external operation on the ethmoid and said it could be done with less bleeding.

T. B. Layton said there were two kinds of sinusitis; one produces a purulent reaction and the other a polypoid reaction. Drainage was the mark of success in the first; it was quite useless in the second.

Walter Howarth said there were two types of sinusitis amenable to treatment; one could be treated by the intranasal method, but in the other, a more advanced type, the intranasal operation had been a failure.

Bedford Russell stated that one serious criticism of the transantral approach was that it was inadequate in scope.

CAMPBELL, Philadelphia.

ZINC IONIZATION OF THE NOSE. ANGELL BUSTILLOS, Arch. venezol. soc. de oto-rino-laring., oftal., neurol. **2**:99 (June) 1941.

Bustillos reported 20 cases of various forms of rhinitis in which he employed the zinc ionization therapy. He states that his is the first attempt to employ this therapeutic measure in Venezuela. The forms treated included simple hypertrophic (congestive) rhinitis, vasomotor rhinitis, hypertrophic rhinitis with degeneration of the mucous membrane, atrophic purulent rhinitis and true atrophic rhinitis.

After outlining his technic in detail, Bustillos summarized the results in his cases and concluded that the simple hypertrophic rhinitis responded most readily to this form of treatment, while the most unfavorable results were obtained in cases of the true atrophic form. Of the 20 cases, definite cure was obtained in 8, improvement in 11 and complete failure in 1. (Abstracter's note: This report is too limited and does not include a sufficient number of cases in each group, nor is the follow-up observation in each case adequate to warrant recommendation for the method of treatment.)

PERSKY, Philadelphia.

Miscellaneous

REMOVAL OF LONGITUDINAL SINUS INVOLVED IN TUMORS. WALTER E. DANDY, Arch. Surg. **41**:244 (Aug.) 1940.

The neurosurgeon is frequently faced with the problem of a tumor of the brain, always a dural meningioma springing from the falx near the midline, which has invaded the longitudinal sinus. Complete removal of the tumor demands ligation and excision of that part of the sinus involved by the tumor. Interference with the blood supply of the cerebral hemispheres, with serious neurologic sequelae, can result. However, in dealing with bilateral tumors, and frequently with unilateral tumors, the longitudinal sinus is often occluded either by compression of the tumor or by direct invasion. Consequently, removal of a section of the sinus adds little or nothing to the demand for a collateral venous circulation. In these cases the venous obstruction has doubtless been gradually progressive, and because of this there has been time for the collateral circulation to develop. Whether a patient's sinus could be similarly resected can only be conjectured; there is no evidence from the literature to support or refute such a claim.

Dandy has reviewed the literature and discovered 9 cases of resection of the longitudinal sinus. In 3 of these cases the resection was made anterior to the rolandic vein. In 1 instance a posterior section of the longitudinal sinus, together with a part of one transverse sinus, and the tentorium were removed. The author adds 4 cases from his own experience. In 2 of these resection was made in the most anterior part of the sinus and in 2 posterior to the rolandic vein. In 2 of the cases the sinus was excised when the tumor had recurred, and in 2 the sinus was removed at the first operation, when the tumor was known to be bilateral. In all of these cases in which occlusion of the sinus had occurred prior to resection no neurologic signs appeared which could be attributed to this important step in

the operative procedure. But there is no available evidence by which it can be known whether the longitudinal sinus can be removed in part before gradually progressive occlusion has occurred.

GRANT, Philadelphia [ARCH. NEUROL. & PSYCHIAT.].

BUCCAL NEURALGIA: A FORM OF ATYPICAL FACIAL NEURALGIA OF SYMPATHETIC ORIGIN. FREDERICK LEET REICHERT, Arch. Surg. **41**:473 (Aug.) 1940.

Buccal neuralgia, a form of atypical facial neuralgia, is characterized by a burning, boring, aching, throbbing pain in the region of the lip, cheek, gum, tongue, maxilla, nose and upper and sometimes lower jaw, spreading at times to the zygoma, into or behind the eyeball or into the temporal region. Anatomic dissection has demonstrated sympathetic fibers from the carotid sinus following the arborization of the facial artery and vein. Extraction of teeth and injection of alcohol into branches of the trigeminal nerve give no relief.

Reichert reports on a series of 30 patients suffering from buccal neuralgia. Tic douloureux of one or more branches of the trigeminal nerve was associated with buccal neuralgia in 10 patients. Three patients with buccal neuralgia had hypothyroidism. The neuralgia was relieved by administration of thyroid extract and vitamin B. Buccal neuralgia was cured in 5 of 8 patients in whom the cervicothoracic portion of the sympathetic chain was interrupted. It was relieved in 13 of 17 patients by the simpler procedure of division of the sympathetic fibers with the facial artery and vein at the lower border of the mandible.

GRANT, Philadelphia [ARCH. NEUROL. & PSYCHIAT.].

TREATMENT OF PNEUMOCOCCIC MENINGITIS. RICHARD G. HORSWELL, WILLIAM H. REALS, WAYNE W. FOX, PAUL S. RHOADS, ARCHIBALD L. HOYNE and BENJAMIN LEVIN, J. A. M. A. **115**:917 (Sept. 14) 1940.

In 22 consecutive cases of pneumococcic meningitis treated with type-specific serum and sulfanilamide or sulfapyridine there were 7 recoveries. There were only 2 recoveries at the Cook County Hospital in the years previous to the introduction of chemotherapy. Pneumococcic meningitis has become more common there than meningococcic meningitis. As soon as the diagnosis of meningitis is made, administration of sulfapyridine or of its sodium salt is started, and the organism is identified or typed as soon as possible. Then the serum is administered intravenously. The concentration of the drug in the blood is checked, and anemia and leukopenia are treated by blood transfusion. Surgical drainage of foci of infection is mandatory, and patients with complications have a poorer prognosis.

HEERSMA, Kalamazoo, Mich. [AM. J. DIS. CHILD.].

SPEECH STUDIES IN CRETINS. S. L. SCHREIBER, I. P. BRONSTEIN and A. W. BROWN, J. Nerv. & Ment. Dis. **72**:169 (Aug.) 1940.

Thirty-two cretins were studied in the clinics of the University of Illinois College of Medicine and the Institute for Juvenile Research in Chicago. The object of the investigation was: (*a*) to determine the extent of the speech sounds of a group of thyroid-deficient children, (*b*) to determine the nature of their speech disorders, (*c*) to compare the speech sounds of this group with those of normal children of corresponding age, (*d*) to compare the extent of the speech disorder of each individual subject with that of the other members of his group, (*e*) to compare the speech disorders of the group as a whole with those of normal children, (*f*) to establish the relation of the children's speech to their intelligence quotients and (*g*) to determine the effect of continuous therapy on the children's speech.

The technical procedures used are discussed and the results tabulated. Only 1 child responded to all of the ninety-eight sounds. No child was able to reproduce all of the sounds correctly. The relation of age and intelligence quotient to the various errors (omission, substitution, approximation and prolongation) showed

that most of the errors occurred at the lower chronologic and mental age levels. Some of the cretins developed language facilities in the short span of six weeks. This phenomenon is attributed to the sudden release of the speech mechanism, due, possibly, to the enhanced basal metabolic rate incident to therapy.

The authors conclude that cretins are able to express the majority of elemental sounds, their speech ability being at the 4 to 5 year old level.

JAHR, Omaha [AM. J. DIS. CHILD.].

THE TREATMENT OF MENINGITIS DUE TO HAEMOPHILUS INFLUENZAE (PFEIFFER'S BACILLUS): A REVIEW OF ONE HUNDRED AND EIGHT CASES. JANVIER W. LINDSAY, E. CLARENCE RICE and MAURICE A. SELINGER, J. Pediat. **17**:220 (Aug.) 1940.

"Meningitis due to H[aemophilus] influenzae is relatively common in children. Excluding tuberculous meningitis, it is the commonest cause of meningitis in children under 2 years of age. . . .

"The treatment of this type of meningitis with specific serum and human complement intrathecally, intravenous serum, with adequate supportive treatment consisting of transfusions, fluids and vitamins, has reduced the mortality percentage from 97 to 83. By adding sulfanilamide and sulfapyridine [2-(paraaminobenzene-sulfonamido)-pyridine] to the above, the mortality for thirteen cases has been further reduced to 54 per cent." RAUH, Cincinnati [AM. J. DIS. CHILD.].

CONDUCTION OF THE ELECTRIC CURRENT IN THE ANIMAL BODY. O. GOEBEL, Monatschr. f. Ohrenh. **74**:425 (Sept.) 1940.

After a lengthy discussion of the physical aspects of electricity and its conduction, it is assumed that the electric current in the body is a free electron current, as in metals. This theory is supported by the existence of direct transmission of radio current onto the cochlear nerve without an ear phone or a loud speaker if the transmission takes place from ear to ear directly. A case is cited in which a girl 10 years of age was able to reproduce thoughts of a person standing close to her.

LEDERER, Chicago.

SYNDROME OF BLUE SCLERA: ITS PRESENCE IN FIVE GENERATIONS. F. CHIMANI, Monatschr. f. Ohrenh. **74**:548 (Nov.) 1940.

The observations on a girl aged 18, evidencing all signs of the syndrome of blue sclera, namely, fragility of the bones and deafness of an otosclerotic type, made possible the study of her family tree. The patient had had several fractures; so had her twin sister and her mother. All of them had blue scleras, but the twin sister showed only slight impairment of hearing. The mother evidenced serious loss of hearing, which had increased with each succeeding pregnancy. A fourth symptom purportedly belonging to this syndrome, a special configuration of the skull, could not be observed. "Rheumatic" joint pains in cases of this condition are explained by the traumatic influence on the joints.

From his study of the family tree, shown in graphic form, and from a survey of the available literature, Chimani concludes that the syndrome of blue sclera is predominantly inherited, both sexes being subject to it and both passing it on alike. Only manifest carriers are capable of transmitting the condition to their offspring. He mentions several theories as to its origin, such as avitaminosis, syphilis and hypofunction of the parathyroid glands. In his opinion, the entity should be placed in the category of a congenital (inheritable) deficiency of the mesenchyme. He further ventures the opinion that otosclerosis, being associated with this syndrome, might not altogether be a disease per se but a part of this systemic disturbance. This is especially evident in some family trees in which the otosclerotic component of the syndrome has become predominant.

LEDERER, Chicago.

REDUCTION OF INTRACRANIAL PRESSURE AFTER OPERATION ON THE BRAIN: PATHOPHYSIOLOGY OF THE CEREBROSPINAL FLUID SYSTEM. H. SPROCKHOFF, Nervenarzt **13**:34 (Aug. 15) 1940.

Sprockhoff points out that in some patients who have undergone surgical operation on the brain the formation of cerebrospinal fluid does not keep pace with the resorption and the result is deficiency and hypotension of the fluid. He lists 11 cases in which there was such postoperative hypotension. With the exception of 2 who had undergone removal of the squama occipitalis, all the patients had a supratentorial defect of the cranium. The patients have headache and are restless and irritable before the hypotensive state develops. The sensorium becomes dulled; slight rigidity of the neck, fatigue, lassitude and nausea develop. There are usually mild hyperthermia and hyperpnea, but the blood pressure and the pulse remain normal. Pallor is occasionally seen. If there is a cranial defect, retraction is noticeable. The pupils become rigid, and reflexes are abolished. Respiration becomes snoring and approaches the Cheyne-Stokes type. This comatose condition may also develop without the prodromal signs. The hypotensive condition does not always lead to complete coma. Differentiation from increased intracranial pressure (brain swelling, arachnoiditis) may be difficult, particularly if there is no cranial defect. The proof that the clinical symptoms described are produced by hypotension in the fluid space is furnished by the fact that intraspinal administration of physiologic solution of sodium chloride promptly counteracts the acute symptoms. If too much fluid is introduced, the skin over the cranial defect will bulge and the undesirable tension may have to be counteracted by another puncture. On the other hand, the retraction in the defect may recur after the filling, so that intraspinal injection has to be repeated. This instability shows that the lack of fluid is not the only cause but that there is also a disturbance in the dynamic equilibrium between formation and absorption of the cerebrospinal fluid. For this reason filling of the fluid space is not sufficient. As few punctures as possible should be made, because every puncture that is not required to counteract acute hypotension or excess pressure retards the regulation. It is necessary to aid the organism in maintaining normal pressure. Fluid should be given in generous quantities—by mouth in the mild form and by intravenous infusion of solutions of sodium chloride or dextrose in the severe form. The author supported the oral administration of fluid with caffeine in order to increase the blood perfusion of the choroid plexus and thereby stimulate the production of the cerebrospinal fluid. Since erect posture promotes absorption of the cerebrospinal fluid and favors decrease in pressure, this posture should be avoided in cases of hypotension, contrary to the general rule in postoperative treatment. Patients with hypotensive conditions complain of headaches which do not differ from those caused by increased intracranial pressure except that they cannot be improved by the withdrawal of spinal fluid but require intraspinal administration of fluid. The prolonged headaches following lumbar puncture complained of by psychopathic persons or by those with a weak sympathetic nervous system are probably of the hypertensive type. Two of the author's patients had chronic hypotensive symptoms and suffered for years with severe headaches. Both had considerable defects in the cranium, and, having noticed that greater severity of headaches was accompanied by greater retraction, they requested plastic repair, after which they were free from complaints. Reduced secretion is the decisive factor, and this in turn must be due to impairment of the plexus. Various causes, such as senile involution, diffuse atrophy of the brain, prolonged existence of internal hydrocephalus, reduced blood pressure and general exsiccation, may be responsible. The author points out that some employ an exsiccating preoperative treatment in order to avoid postoperative swelling of the brain. This preoperative exsiccation proved its value. The author does not reject the method but emphasizes that it should be used with discrimination, for if it is employed in all cases postoperative hypotension will be more frequent. It should not be employed in cases in which the previous history or the clinical aspects indicate a tendency to hypotension.

J. A. M. A.

Society Transactions

COLLEGE OF PHYSICIANS OF PHILADELPHIA, SECTION ON OTOLARYNGOLOGY

KARL M. HOUSER, M.D., *Chairman*

WILLIAM HEWSON, M.D., *Clerk*

GEORGE L. WHELAN, M.D., *Editor of Abstracts*

Regular Meeting, Feb. 18, 1942

Paralysis of the Larynx. DR. LOUIS H. CLERF.

In a study of paralysis of the larynx it is important to recall that two main actions of the larynx are separation and approximation of the two vocal cords. This is accomplished by two groups of opposing muscles, namely, the abductors and the adductors. These muscles receive their motor impulses from a common nerve, the inferior, or recurrent, laryngeal branch of the vagus nerve.

Abduction, or separation, of the vocal cords is accomplished by the posterior cricoarytenoid muscles. These rotate the vocal process of each arytenoid cartilage outward and separate the vocal cords. The principal adductors are the lateral cricoarytenoid muscles, which rotate the vocal process of each arytenoid cartilage inward, approximating the membranous cords, and the interarytenoid muscle, which approximates the two arytenoid cartilages. The cricothyroid and thyroarytenoid muscles also aid in adduction, but primarily they are tensors of the vocal cords.

Section of a recurrent laryngeal nerve is promptly followed by almost complete adduction of the corresponding vocal cord. The muscles of the opposite side of the larynx remain unimpaired, and there is loss of tension of the membranous portions. Tension of the cord is maintained by the cricothyroid muscle, which also exerts some adductor action. This muscle is supplied by the superior laryngeal nerve. The thyroarytenoid muscles also exert some effect on tension, as well as on adduction.

In the early stages unilateral paralysis of the larynx usually produces slight voice disturbances. These are dependent almost entirely on the degree of loss of tension. A certain number of patients with unilateral paralysis also have difficulty with swallowing, owing to interference with the protective function of the larynx. Rarely does one observe dyspnea. Bilateral paralysis of the larynx commonly is associated with dyspnea, unless there is considerable loss of tension of the vocal cords. The voice disturbances are dependent on the degree of loss of tension.

Many operative procedures have been used for the relief of dyspnea in cases of bilateral paralysis of the larynx. The majority of these methods have not been satisfactory, owing to the recurrence of the stenosis, which commonly assumed a cicatricial, as well as a paralytic, character. The outstanding procedure to date is the King operation, which as practiced originally had for its purpose immobilization of the arytenoid cartilage and implantation of the omohyoid muscle to the vocal process of this cartilage. Recent observations indicate that this muscle probably exerts no remarkable influence and the beneficial effects are due to displacement outward and fixation of the arytenoid cartilage. This structure usually remains fixed, and narrowing of the glottis during phonation is dependent on movement of the intact side of the larynx.

The results of the King operation in a series of 7 cases are reported. In 3 cases the results were not satisfactory, owing to failure to fix the arytenoid

cartilage laterally. In 2 of the 3 cases a second operation has since been done and an excellent result secured. In the remaining 4 cases satisfactory results were obtained by a single operation. In 1 case the omohyoid muscle, which had been cut at the time of the thyroidectomy, could not be utilized. The operation consisted only of mobilization of the arytenoid cartilage, which was then fixed laterally to the thyroid cartilage. All of the patients have dispensed with the tracheotomy cannula and have a satisfactory airway. There is some weakness of the voice, but this is not great, and ultimate functional results have been satisfactory.

Criteria for the Selection of Treatment of Cancer of the Larynx. Dr. Chevalier L. Jackson and Dr. John V. Blady (by invitation).

This paper will be published in full in a future issue of the Archives.

Benign Tumors of the Larynx: Inflammations and Neoplasms. Dr. Gabriel Tucker.

In the larynx a tumor histologically benign may prove fatal by obliterative pressure, paralyzing pressure or massive obstruction. It may recur persistently, but the recurrence is a new process, not an infiltration of the adjacent normal cells. The growth does not metastasize. Histologists try to reserve the ending oma for true neoplasms, although the Greek suffix, meaning tumor, does not justify the limitation and the distinction in many cases is impossible. One cannot estimate the frequency of laryngeal tumors from the literature because only an infinitesimal number of such lesions are reported, or even diagnosed. When one tries to determine the relative frequency of the different kinds of growths, one is confronted with confusion, for histologic nomenclature has depended on the varying viewpoints of individual histopathologists. For example, there is no uniformity of opinion as to how much fibrous tissue a growth must show to be called a fibroma. What one histologist calls a fibroma another reports as chronic inflammatory tissue. Therefore statistics on incidence are worthless.

The cause of true neoplasms is unknown. The etiology of the tumors of inflammatory origin is involved in the manifold meshes of the network of causes of chronic laryngitis. Some tumors, such as hematoma and organizing hematoma, are not primarily inflammatory. In the earlier days of cellular pathology histologists drew a sharp line between inflammatory and neoplastic processes, and even today they try to distinguish between inflammatory growths and true neoplasms. Yet no one can maintain that inflammation does not enter into the cause of many true neoplasms; any neoplasm, even carcinoma, which of all growths is one of the most typically neoplastic, is certainly in some cases caused in part by trauma, irritation, inflammation and frustrated repair. Every surgeon advises the removal of a benign growth subject to irritation lest it become malignant. Clinically, however, the laryngologist is confronted with the necessity for decision as to the probable benignancy or malignancy of any growth, for this decision is the basis of the surgical treatment. The causes of benign true neoplasms are unknown; probably trauma, in its widest sense, is a factor. Certainly, in a large group of benign tumors and tumor-like formations inflammation is the chief etiologic factor. All forms of vocal abuse are, essentially and etiologically, forms of trauma; so are tobacco smoke and dust. Fumes, irritating vapors and war gases are certainly factors in the causation of tumors. [Color cinematograms of the various benign tumors of the larynx were presented.]

The Larynx, Tracheobronchial Tree and Esophagus in Kodachrome. Dr. Paul H. Holinger, Chicago.

Photography serves two principal purposes in medicine: first, to illustrate the normal and pathologic appearance of various organs or parts of the body, and second, to record the clinical progress in a case or a series of cases. By means of motion pictures taken through the laryngoscope, the bronchoscope and the

esophagoscope, it is now possible to extend the field of medical photography to the formerly inaccessible regions of the lower air and food passages and to record the anatomy and pathology of these areas in color.

The camera devised for endoscopic photography consists of a magazine-loading, 16 mm. camera and a f 2.7 3 inch (7.5 cm.) lens. Illumination of the small endoscopic field is obtained by directing the image of the ribbon of a ribbon filament projection lamp onto a 45 degree mirror, which encroaches on the lumen of the endoscope. This type of axial illumination provides adequately the illumination essential to color photography. A specially constructed telescope inserted into the system permits visualization of the field being photographed, not only during the finding but during the actual filming.

The Kodachrome films shown illustrate the indirect (mirror) appearance of the normal larynx, as compared with the direct view obtained through the laryngoscope. They also illustrate such pathologic lesions of the tracheobronchial tree and esophagus as bronchiectasis, pulmonary adenoma, bronchial tuberculosis, bronchogenic carcinoma, cardiospasm and carcinoma of the esophagus. The endoscopic appearance and mechanical problems of foreign bodies in the air and food passages are likewise illustrated by photographs of various foreign bodies introduced into the larynx, bronchi and esophagus of experimental animals.

KARL M. HOUSER, M.D., *Chairman*

WILLIAM HEWSON, M.D., *Clerk*

GEORGE L. WHELAN, M.D., *Editor of Abstracts*

Regular Meeting, April 15, 1942

MEDICAL AND SURGICAL INDICATIONS IN OTOLARYNGOLOGY

Allergic Aspects. DR. MERLE M. MILLER.

In discussing allergic diseases from the standpoint of the nose and throat, one must consider the following conditions: (1) uncomplicated seasonal nasal symptoms; (2) perennial nasal symptoms, both with and without infection, and (3) the nasal symptom complex occurring with urticaria, angioneurotic edema, migraine, gastrointestinal allergy and other so-called allergic manifestations. The relation of nasal allergy and sinal infection to bronchial asthma, or the role of pathologic or pathophysiologic nasal conditions as a factor in bronchial asthma, must also be evaluated.

Before attempting to discuss in detail the management of the aforementioned conditions, it may be well to review briefly the factors to be considered in routine diagnosis in a case of suspected nasal allergy.

1. Hereditary history of allergy. Estimations of the incidence of nasal allergy vary from 40 to 80 per cent of patients with a direct family background of allergy.

2. Presence or history of associated allergic manifestations in the patient.

3. Physical abnormalities, especially an endocrine dysfunction.

4. Results of laboratory examinations, including blood count, Wassermann test, chemical studies of the blood and urinalysis. With the possible exception of eosinophilia, there is no laboratory evidence that is diagnostic of allergic disease.

5. Nasal symptoms—sneezing, itching, burning, discharge and block.

6. Signs in the nose—obstruction, edema and color of the membranes.

7. Roentgenographic evidence of involvement of the sinuses—edema, polyps, retained secretions.

8. Character of nasal secretions—color, consistency, p_H and cytologic features.

9. Associated conditions of the eyes, ears and throat.

10. Concurrent allergic disease, such as asthma, hay fever, urticaria and eczema.

11. Cutaneous reactions to scratch, intracutaneous, patch and passive transfer tests. Any discussion of seasonal hay fever can readily be concluded, as at present it is the only allergic disease for which cutaneous testing has any degree of accuracy. Also, a fairly satisfactory number of patients respond to hyposensitization. Some patients with hay fever do not respond and are often extremely refractory to treatment. It is possible that in the study of the molds, smut and rust content of the air, light will be thrown on complicating seasonal factors.

The diagnosis of perennial nasal manifestations is usually much more involved and indefinite than that of seasonal hay fever. It is here that cytologic studies of the nasal secretions may give definite information about the etiologic factors concerned.

Treatment of nonseasonal nasal block, rhinitis and rhinorrhea is also discouraging at times. From the standpoint of infection, the otolaryngologist is qualified to treat the patient adequately. From the allergic side, all are at times completely baffled. At present, allergists still continue to employ elimination and hyposensitization. Enthusiasm, as well as pessimism, is evidenced among allergists when exceptionally good results are claimed.

There is a considerable difference of opinion as to the cause of polyps, and many suggestions have been made as to their classification. General constitutional predisposition and heredity are thought by some to play a part. Chronic catarrhal inflammation of the ethmoid cells and maxillary antrums is also designated as an etiologic factor. Not in agreement with the theory of infection is a group who believe that all polyps have an allergic basis.

I do not have time here to go into the histopathology of nasal polyps, even though it is important. In passing, I may state that in all cases edema is present and that there are thickening of the basement membrane and in most cases infiltration of eosinophils, together with lymphocytes and other cells. Of utmost importance is the fact that in 50 to 65 per cent of cases there is localized or pronounced fibrosis.

The treatment of polyps can be outlined as follows: (1) elimination of chronic infection; (2) allergic therapy—elimination and hyposensitization—and (3) removal, if there is obstruction, and especially if the lesion is of long standing and fibrotic. Although some men believe that polyps will disappear as soon as treatment of the allergic condition is instituted, this opinion is open to doubt. Both the type of polyps and their position influence the choice of therapeutic measures. If polyps have to be removed, it is best to refrain from surgical intervention during the pollen season.

The tonsils may be the foci of infection in cases of bronchial asthma. After removal the majority of patients are relieved. Symptoms may return. Most patients, however, have been greatly benefited by tonsillectomy.

The diagnosis of chronic suppuration of the paranasal sinuses should never be based on the evidence of chronic nasal discharge or on positive roentgenographic signs alone. Hyperplastic, infectious sinusitis must be differentiated from the edematous, polypoid, so-called allergic type.

If chronic infection persists, if the mucosa of the maxillary sinuses is greatly thickened or if there is confined pus, surgical intervention is necessary. Too much dependency should not be placed on allergic therapy; however, in selected cases it is worthy of a trial.

Pathologic Aspects. DR. JOHN EIMAN (by invitation).

Acute Addisonian Syndrome Precipitated by Sphenoiditis Due to Hemolytic Streptococcus.

S. R., a white man aged 63, was admitted to the Abington Hospital on Dec. 27, 1937 in coma, with the diagnosis of uremia. Urinalysis revealed no albumin or casts. Chemical studies of the blood gave the following results:

nonprotein nitrogen, 13 mg. per hundred cubic centimeters; carbon dioxide-combining power, 46 volumes per cent; sugar, 83 mg. per hundred centimeters; chlorides, 430 mg. per hundred cubic centimeters. The chlorides of the urine measured 970 mg. per hundred centimeters. Obviously, this case was not one of uremia. A diagnosis of addisonian syndrome was made because the chloride content of the plasma was 430 mg. and that of the urine at the same time was 970 mg. per hundred cubic centimeters.

Autopsy showed atrophy of the adrenals, due to lack of adrenotropic hormone of the pituitary. The pituitary was transformed into a large cyst, with only small numbers of active cells in the wall. This small number of pituitary cells apparently had been enough to supply the basic needs of pituitary secretion. This supply, however, had been cut off rather suddenly. Cultures of material from the sphenoid sinus showed hemolytic streptococci.

Anthrax with Primary Lesion in the Nares: Report of a Case.

W. H. S., a white man aged 71, a maintenance man in a leather factory, complained of weakness two days before admission to Dr. F. Krauss's service, Abington Hospital. The next day he went to work but noticed dryness of the nose and throat. In the afternoon he thought that he had acquired a cold in the head. In the evening his nose was discharging clear fluid, and he had a frontal headache. The following morning there were swelling of the right side of the neck and a sensation of fulness in his throat. He had slight difficulty in breathing and swallowing.

On examination both nasal passages were found to be congested and occluded. The right side showed sanguineous, mucopurulent discharge; the left, mucopurulent discharge. The mucosa was red. There was no paranasal tenderness. The tonsillar pillars, the soft palate and the posterior pharyngeal wall were injected and edematous.

Cultures of the nasal discharge showed anthrax bacilli in pure culture. The blood culture showed 30 organisms per cubic centimeter.

Notwithstanding the administration of large doses of antianthrax serum and sulfathiazole (2-[paraaminobenzenesulfonamido]-thiazole) the patient died four days after admission. The cause of death was bacteremia and edema of the glottis.

Acute Laryngitis Due to Haemophilus Influenzae.

J. T., a white girl aged 3 years, was admitted to the Abington Hospital on Dec. 3, 1938. At 3 a. m. she complained of pain in the throat, and at 6 a. m. she vomited and was sent to the hospital. On admission the posterior pharyngeal wall showed pronounced inflammatory edema. The child was in acute respiratory distress and was cyanotic. Tracheotomy was done, with immediate relief.

Cultures of material from the larynx and of the blood showed H. influenzae. The patient was given 20,000 units of anti-H. influenzae type B. serum. She recovered.

Sinclair reported 10 similar cases (Haemophilus Influenzae Type B in Acute Laryngitis with Bacteremia, *J. A. M. A.* **117**:170 [July 17] 1941), with 4 deaths and 6 recoveries. He employed drugs of the sulfonamide series in all cases and anti-H. influenzae type B rabbit serum in only 2 cases. Tracheotomy was performed in 6 cases.

Meningitis Complicating Infection with Hemolytic Streptococcus.

A case of mastoiditis followed by meningitis caused by strictly anaerobic hemolytic streptococci was reported by the late Dr. Fred Smith and associates (Anaerobic Beta Hemolytic Streptococcus Meningitis of Otitic Origin Treated with Sulfanilamide and Culminating in Complete Recovery, *J. A. M. A.* **110**:887 [March 19] 1938). The patient recovered after the use of sulfanilamide and azosulfamide.

Most hemolytic streptococci will grow on blood plates under ordinary aerobic incubation. There are, however, many strains of hemolytic streptococci and other pathogenic bacteria which will not grow at all, or very sparingly, under aerobic conditions but will grow readily, even luxuriantly, in a "candle jar." In

the candle jar there is only a slight reduction of oxygen. There is, however, definite increase in carbon dioxide, up to 3 per cent. Fildes demonstrated that many organisms could be grown more successfully in an atmosphere of increased carbon dioxide (*Brit. J. Exper. Path.* **16**:309 [June] 1935). The candle jar method was first employed by Parker and Hudson (*Am. J. Path.* **2**:357 [Sept.] 1926). My colleagues and I find that by the routine use of the candle jar we have increased the recovery of pathogenic organisms in general and the streptococci in particular.

Otitis Media Due to Hemolytic Streptococcus Followed by Meningitis.

H. S., a Negro boy aged 11 years, recovered from hemolytic streptococcus meningitis following otitis media with use of sulfadiazine (2-[paraaminobenzenesulfonamido]-pyrimidine).

It is of importance to note that when one of the sulfonamide compounds (at present sulfadiazine) is used in treatment of meningitis, it should be given in large enough doses to raise the concentration of the drug in the blood and the spinal fluid to effective levels. The case of R. P., a white boy aged 19 months with influenzal meningitis, illustrates this point. The spinal fluid did not become sterile until the dose of sulfadiazine was raised to 8 Gm. per day and the concentration in the spinal fluid reached 20 mg.

DISCUSSION

DR. KARL M. HOUSER: I have been using the autofiltrates in treating those nasal conditions recognized as hypersensitive states and amenable to such treatment, for example, hay fever, vasomotor rhinitis and even acute coryza. I should like to know whether Dr. Eiman approves of this therapy.

DR. JOHN EIMAN: The use of autofiltrates meets entirely with my approval. Unfortunately, not enough work has been done on this therapy. There is much that is not known about these substances; success in the treatment of intranasal diseases with autofiltrates made from the nasal secretions probably depends on the proper regulation of the dose. Here, again, there is a wide divergence of opinion. One also must consider whether or not the condition is acute or chronic.

Hematologic Aspects. DR. MAX M. STRUMIA (by invitation).

In general, infections in the field of otolaryngology may be divided into three types on the basis of the hematologic picture.

1. Minor, relatively superficial inflammatory processes in which as a rule there is little or no change in the morphologic blood picture.

2. Inflammatory processes of greater surface extension, sometimes with ulceration in certain areas but with little penetration of the lesion. In this type, as a rule, the morphologic picture of the blood is only slightly changed. This is particularly true in cases of acute tonsillitis and nasopharyngitis, unless due to a virus. There is, in other words, an apparent disproportion between the clinical picture and the blood picture.

3. Abscess formation or retention of pus, with changes in the picture more or less proportional to the involvement and extent of the lesion.

These three groups have in common the basic fact that the alteration of the blood picture is the result of a toxic factor from an inflammatory process of the upper respiratory system which affects the circulation as well as the hemopoietic centers. However, the otolaryngologist is often called on to attend a patient with an inflammatory process of the upper respiratory tract, particularly of the mouth and throat, which is the result, rather than the cause, of a primary disturbance of the hemopoietic organs. Patients of this type are usually classifiable into two groups: (1) those with the agranulocytic syndrome, and (2) those with acute leukemias.

Finally, one must consider a disease in which, probably, both mechanisms are involved. I refer to acute glandular fever, also called infectious mononucleosis.

In this disease there is at the onset a mild, acute inflammatory process of the throat accompanied by lymphadenopathy, with moderate neutropenia and, as a rule, lymphocytosis, with presence of the typical degenerated forms. In the course of the disease not infrequently the mild, acute inflammatory process of the throat, possibly of virus origin, changes to a more severe, ulcerative, sometimes septic, infection of the throat. This usually is due to secondary infection with pneumococci or streptococci, aided by the existing neutropenia.

Otolaryngologic Aspects. Dr. M. Valentine Miller.

Operation on the frontal, maxillary or other sinuses in the case of an allergic patient should be carried out only after thoroughgoing antiallergic treatment, except, of course, when the need for operation is so urgent as not to permit of delay. With this plan some of the patients, who in the past would have been operated on, may respond in a satisfactory manner to antiallergic treatment, with the result that operation may be unnecessary. This has been found to be true in cases in which a few years ago one would have thought by all the rules that operation should be carried out. Generally, when operation is performed for allergic sinusitis there is cessation of symptoms over a period extending from six weeks to six months, usually midway between, after which the allergic symptoms recur when the patient is exposed to allergens to which he is sensitive. With this fact clearly understood, there is no reason that operation on the sinuses should not be carried out when indicated.

My conception of chronic disease of the nasal accessory sinuses has led me to believe that there are certain patients for whom these essential principles of surgery are not only indicated but undeniably imperative if a cure is to be hoped for. When the lining mucous membrance of a sinus is invaded by fibrous connective tissue, divested of its cilia and glandular elements and damaged by avascularity resulting from endarteritis and thrombophlebitis, the injury is irreparable, and reconstruction and rehabilitation are impossible. Consequently, under such conditions, particularly when anatomic conformations preclude the possibility of one's reaching into the remote recesses of the frontoethmoid cells or the frontal sinuses, it would seem that the sinus, stripped of its diseased mucous membrane, should be exposed widely to inspection. Even when rigid adherence has been given to these immutable principles of sinus surgery, including, perhaps, excision of osteomyelitic walls, it is not always possible to secure a result that is beyond reproach.

News and Comment

CASSELBERRY AWARD OF THE AMERICAN LARYNGOLOGICAL ASSOCIATION

A sum of money is now available from the Casselberry Fund of the American Laryngological Association for a prize award, decoration or grant to be given for original investigation in the art and science of laryngology or rhinology. Theses must be in the hands of the secretary, Dr. Arthur W. Proetz, 1010 Beaumont Medical Building, St. Louis, before March 1, 1943.

Directory of Otolaryngologic Societies *

NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Dr. Claude C. Cody, 1304 Walker Ave., Houston, Texas.
Secretary: Dr. Louis H. Clerf, 1530 Locust St., Philadelphia.

American Academy of Ophthalmology and Otolaryngology

President: Dr. Ralph I. Lloyd, 14–8th Ave., Brooklyn.
President-Elect: Dr. James A. Babbitt, 1912 Spruce St., Philadelphia.
Executive Secretary: Dr. William L. Benedict, Mayo Clinic, Rochester, Minn.

American Broncho-Esophagological Association

President: Dr. Robert L. Moorhead, 125 Ramsen St., Brooklyn.
Secretary: Dr. Paul Holinger, 700 N. Michigan Ave., Chicago.

American Laryngological Association

President: Dr. Charles J. Imperatori, 108 E. 38th St., New York.
Secretary: Dr. Arthur W. Proetz, Beaumont Bldg., St. Louis.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. James G. Dwyer, 375 Park Ave., New York.
President-Elect: Dr. H. Marshall Taylor, 111 W. Adams St., Jacksonville, Fla.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester, N. Y.

SECTIONS:

Eastern.—Chairman: Dr. Edward J. Whalen, 750 Main St., Hartford, Conn.
Place: Hartford, Conn. Time: Jan. 15, 1943.

Southern.—Chairman: Dr. Francis B. Blackmar, Swift Kyle Bldg., Columbus, Ga.
Place: Chattanooga, Tenn. Time: Jan. 28, 1943.

Middle.—Chairman: Dr. Voss Harrell, Hofman Bldg., Detroit.
Place: Detroit, Mich. Time: Jan. 20, 1943.

Western.—Chairman: Dr. Irving M. Lupton, Medical Arts Bldg., Portland, Ore.
Place: Portland, Ore. Time: Jan. 31, 1943.

American Otological Society

President: Dr. Wesley C. Bowers, 17 E. 61st St., New York.
Secretary: Dr. Isidore Friesner, 101 E. 73d St., New York.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.

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